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PHILADELPHIA

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THE LOCALIZATION OF THE HIGHER PSYCHIC FUNCTIONS,

WITH SPECIAL REFERENCE TO THE PREFRONTAL LOBE.*

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PHILADELPHIA.

Cerebral localization had several epochs during the nineteenth century. At its dawn the views of Gall had awakened interest not only in the scientific world, but in all the European centers of population. Gall had boldly advanced the idea that the brain was not only subdivided into centers of special function, but that these were indicated on the outside of the head by peculiar prominences and conformations. An essential element of his phrenology was the existence of cranial salients, recognizable by the skilled fingers of the examiner, and constituting the ectal correlatives of ental physiologic centers in the brain substance. The bumps of the phrenologist represented gyres and subgyres, in which resided, as in the compartments of an elaborately arranged house, the habitants of the soul or mind.

Gall, who was a worker as well as theorizer, eventually lost himself in mazes of his own creation, but he will at least be remembered as one who fastened the eyes of the world on the truth that all parts of the brain do not act together for a single purpose, and that a single part of the brain is not sufficient for all the functions of the mind.

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* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-sixth Annual Session, July, 1905.

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For nearly three-quarters of a century the advances made in our knowledge of cerebral localization were limited. The one great discovery before 1870 was that of the existence of a center for speech in the hinder part of the left third frontal convolution—the center of Broca.

As the result of the impetus given by Hughlings Jackson, in his suggestion that certain small areas of the cerebral cortex were representative of particular movements, the researches of Hitzig and Fritsch, of Ferrier, Charcot and others were given to the world between 1870 and 1880, and the existence of a cortical motor zone with many subareas or centers was soon almost universally recognized. Before long, visual, auditory and other sensory phenomena were found to have their cortical representation.

For nearly a quarter of a century attention was concentrated on the discovery of separate cortical centers, until Flechsig (1893), through his embryologic researches, demonstrated the importance of recognizing not only primordial centers of sense and of movement, but also the existence of cerebral association areas concerned with intermediate and higher psychic functions. Since the period inaugurated by Flechsig, the subject has evolved in two directions: 1, In the direction of a more exact delimitation of areas and centers concerned with primary functions; 2, in that of the recognition of subdivisions of Flechsig's posterior association area, the concrete concept area or the concrete memory field. The visual and auditory subdivisions of this concrete memory field have in particular received some exact subdivision through clinicopathologic, morphologic, anatomic and physiologic research. The views of the authors in regard to cortical localization in general have been indicated in diagrammatic schemes published by one of us.[1] It will be observed in studying these schemes that not only are lower and higher areas of vision and audition indicated, but also special areas for cutaneous and muscular sensibility and stereognostic conception.

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[1] Mills, C. K.: "The Physiologic Areas and Centers of the Cerebral Cortex of Man, with New Diagrammatic Schemes," Univ. of Penna. Med. Bulletin, May, 1904. Also: "Subdivisions of the Concrete Concept Area of the Human Cerebrum," Medical News, Nov. 5, 1905. Also: Mills, C. K. and Frazier, C. H.: "The Motor Area of the Human Cerebrum, Its Position and Subdivisions, with Some Discussion of the Surgery of this Area," Univ. of Penna. Med. Bulletin, July-August, 1905.

Each sensory zone has contiguous to it a motor area or center, the main motor region being that which is closely related topographically, as it is functionally, to the areas of cutaneous and muscular sensibility.

We shall not go into any detailed consideration of these zones, subareas and centers. This glance at the history and the present status of cerebral localization is given in order to lead up to the particular subject of the present paper, which is concerned with the centers of the highest psychic functions. We hold to the view that the highest mental faculties or functions have their material representation in the prefrontal lobes of the brain, and especially in the left prefrontal lobe. Our concern will be to give the general data and reasons for this belief, supplementing the paper with a report of a case of tumor of the left prefrontal lobe recently observed.

Gall and his followers made an elaborate subdivision of the faculties of the mind as understood in their time. One mistake made by them was that they did not recognize the essential difference between a fundamental faculty or function of the brain and certain cerebral side attributes or epiphenomena. The ability to attend, to will, to judge, to compare, to reason and to exercise the imagination are fundamental psychic functions; the exhibition by an individual of pride, of vanity, of friendship, of combativeness, of piety, of filial love, of sexual passion or similar propensities, is not due to the operation of any single mental faculty or function. The former are the vocations, the latter the avocations of the mind. The former are represented by special centers associated by special tracts; the latter result from the action of one or several or many of the regions of the brain.

The evidence afforded by studies in human and comparative morphology and anatomy of the cerebral surface in favor of the view that the prefrontal lobe, and especially the left prefrontal lobe, is the seat of the highest mental faculties, is of much value. Already a considerable number of the brains of notable men presenting special fissural and gyral arrangement and development of the prefrontal region indicative of high or unusual endowment have been put on record. Reference need only be made to the classical cases of Gauss, Helmholtz and Grote and to the more recent studies of E. A. Spitzka on the brains of the two Seguins, Cope, Harrison Allen, Major Powell and others. If the anatomic and morphologic characteristics of such brains are placed side by side with those of criminals, paranoiacs, negroes and other human beings of low individual or racial development (as studied by Dr. Mills and others), the psychophysiologic importance of the prefrontal region becomes apparent. It might be remarked in passing that morphologic and anatomic studies do not always seem to give to the left prefrontal lobe the preponderance in higher mentality attributed to it by some. Spitzka, for instance, found in the brain of Major Powell a preponderant development of the mesal surface of the right frontal lobe. In studies of this kind a comparison should always carefully be made between the fissural and gyral characteristics of the prefrontal and parieto-temporo-occipital areas. A superior development of the latter is not always accompanied by an equally superior pattern of the former. In rare cases the former may largely outclass the latter.

Morphologic studies of the brains of imbeciles, such as have been made by Wilmarth and others, are corroborative of the view of the higher psychic functions of the prefrontal region. These brains are, as a rule, characterized by unusual simplicity in fissural and gyral arrangement.

The embryologic researches of Flechsig and the histologic investigations of Campbell show, among other things, the absence of projection cells and fibers in this portion of the brain. The latter speaks of the prefrontal region as the very last pallium to appear in the progress of phylogenesis. We may accept this statement and regard it as in accord with our views of the functions of this part of the brain. The last and the least organized portion of the nervous system is the most highly organized and most specially endowed.

Gross and microscopic examinations of the brains of general paretics support in some degree the thesis that the prefrontal portion of the cerebrum is the seat of its highest functions. It is probable that careful pathologic investigation of the brains in cases of dementia præcox will afford similar evidence.

Presuming that the prefrontal lobe is especially concerned with the highest intellectual functions, the question of whether or not this region is divided into subareas and centers is one which is naturally suggested. In such a study we have not to guide us comparatively coarse and easily differentiated phenomena-like movements and disorders of cutaneous sensibility, or of the special senses.

The higher psychic faculties themselves are indeed usually so interblended in expression as to make a study of their relative maintenance, loss or increase in particular individuals, a matter requiring trained powers of observation and analysis, such as are not often possessed, or at least exercised, by those recording cases of disease, arrest of development or unusual endowment. It is possible that the clue to the subdivisions of the highest psychic region may be found by a consideration of the position and relations of the centers of known functions contiguous to the prefrontal lobe. Intermediate between this lobe and the area recognized as motor are situated centers concerned with speech and with writing. Broca's center is situated just anterior to the centers for articulation, enunciation and phonation, the subdivisions of the so-called face area. Speech is the chief instrument employed in reasoning and the psychic centers of ratiocination should be topographically closely related in position to the center of Broca. Writing administers to exactness in thinking and the centers of comparison and judgment may have their highest development contiguous to the graphic center. While in the main the motor centers, for the face, arm, trunk and leg are limited to the precentral convolution, the centers for movements of the head and eyes, and especially for movements of the eyes alone, are thrust toward and into the prefrontal lobe. Attention and volition have their most marked physical expression through vision and position of the head, and if centers for these mental faculties or processes are differentiated their most probable situation is in parts contiguous to centers for movements of the eyes and head.

One of the greatest difficulties of proving the localization of the highest intellectual processes has arisen from the fact that physicians, even those trained in neurology and psychiatry, frequently fail to note the mental symptoms carefully or describe them in such an uncertain manner as to make the records of little value. It is surprising, considering the richness and profusion of the clinicopathologic contributions to cerebral localization during the last two or three decades, how few of these have any notable value regarding the higher psychic functions. In our own language, the contributions of Phelps[2] are of especial value. An elaborate study of personal and other cases is made by Phelps, who arrives at the conclusion that the left prefrontal lobe is the seat of the highest mental processes. He says:

> The large number of cases cited, with the analysis of their symptoms and lesions, are probably sufficient in themselves to form a basis for conclusions. They represent the personal observation and record of 800 cases of intracranial traumatism, of which more than 300 were subjected to either operative or postmortem inspection. Excluding those cases in which death had been preceded by primary and permanent unconsciousness, they were all germane to the present inquiry as showing either the presence of left frontal lesion where mental symptoms had been noted, or the absence of such symptoms where the lesion was situated in any other region of the brain.

Besides the series of traumatic cases compiled by Phelps, other collections of cases of prefrontal lesion have from time to time been published, as well as a considerable number of isolated cases either due to disease or injury. Some of these, like the crow-bar case, have so often been recalled that they are now well known to the profession at large. The inferences drawn from both the individual cases and the compilations have been conflicting, some holding that they prove nothing as regards the functions of this portion of the brain or only indicate that it is physiologically negative; on the other hand, a very considerable number of cases of lesions situated elsewhere than in the prefrontal lobes and giving symptoms indicative of the involvement of the higher psychic faculties, have been recorded. We believe, however, that the comparatively conflicting cases can be satisfactorily explained. In many instances the individuals whose cases are reported have been of a relatively low intellectual status, and as already intimated, physicians in general are not well equipped to put on record psychic symptoms in detail. Frequently the mental condition and symptoms, which are due to the irritative or distant effects of a cerebral lesion, especially when this is a tumor are not taken into account by the reporter. We believe that if each case of prefrontal disease and especially destructive disease of the left prefrontal lobe, is carefully studied it will be found that mental symptoms of a special character can always be recognized, and that these will be in the domain of the higher psychic activities.

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[2] Phelps, Charles, New York Med. Jour., Nov. 10, 1894, to Jan. 12, 1895; "Traumatic Injuries of the Brain," etc., New York, 1897, 1900; Amer. Jour. of the Med. Sci., April, 1902.

E. Müller, in 1902, published three exhaustive papers on the frontal lobe, his chief object in these contributions being to present the question of the psychic functions of this portion of the brain. His frontal lobe practically coincides with the prefrontal lobe of the writer's—that portion of the brain in advance of the generally accepted motor region and of the graphic and motor speech centers. While he gives details of only one case of his own, he has collected from the literature no less than 164 cases of lesion or disturbance of this portion of the brain, these including tumors, concussion, injuries and other lesions.[3]

We have only space in this paper to give his most important conclusions, with brief references to one or two points of special interest in his studies, which were not only clinicopathologic, but included to some extent a consideration of facts of physiology, morphology and psychiatry. He asserts, as the result of his studies, that tumors of the frontal lobe need not always cause disturbance of the higher psychic functions, but admits that they do this frequently. He holds that psychic disorders of various sort may result from tumors situated in any portion of the brain. He believes, further, that the disorders of the higher intellectual functions which are so frequently noted in prefrontal lesions, are due, especially in the case of tumors, to the fact that these being in a region latent as regards physical symptoms, the cases remain a longer time under observation, and hence the mental disorders are more frequently studied. He contends that psychic symptoms in cases of brain tumor do not occur alone, but always in association with other symptoms. He does not believe that prefrontal tumors necessarily produce psychic symptoms of a special kind. He does not believe that morbid wittiness (Witzelsucht), which has been advanced by some German writers as a symptom especially depending on disease of the frontal lobes, is to be attributed to lesion or disturbance of this region, his analysis of cases seeming to show that it may occur from lesions in various parts of the brain. In 22 cases of tumors or other lesions involving both frontal lobes cited by him, this symptom was absent. We might remark in passing that although we have seen many cases of brain tumor, a number reaching into the hundreds, this symptom has been extremely rare in our experience.

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[3] Deutsche Zeitschrift für Nervenheilkunde, vol. 21, 22, 1902; Allgemeine Zeitschrift für Psychiatrie, vol. 59, 1902.

Schuster,[4] also in 1902, contributed a paper based on a study of no less than 775 cases of tumor. He found from his analysis that tumors of the callosum always gave mental symptoms; next in order came tumors of the frontal lobes, which gave the same symptoms in 80 per cent. of the cases; tumors of the temporal, parietal and occipital lobes gave psychic disorders in from 54 to 66 per cent., while tumors of the base gave these symptoms in only 25 per cent., and cerebellar tumors in 35 per cent.

Both Müller and Schuster agree that in the majority of instances the prevailing psychic disorders are disturbances of the sensorium, of orientation and of consciousness, the last showing themselves chiefly in hebetude or stupor.

Von Monakow,[5] in a paper published in 1904, refers to the statistics of Müller, Schuster and others, and discusses the subject from a physiologic point of view, concluding that the physiologic evidence is insufficient to prove that the psychic functions are especially located in the prefrontal portion of the brain. He believes that psychic disturbance may occur from tumors situated anywhere in the cranial cavity. He also agrees with Müller and Schuster that tumors of the prefrontal area need not cause disturbance of the higher psychic functions.

The papers of Müller, Schuster and Von Monakow, like many others of less elaboration written on this subject, are defective in their discussion of the subject, in that clear distinctions are not made as to the character of the psychic disturbances caused by tumors located in various parts of the brain. The chief sources of fallacy would seem to be (1) that sufficient stress is not laid on the fact that owing to the painful and exhausting character of brain tumors, mental disturbances in the broad sense may occur because of the irritation and exhaustion produced by these lesions; (2) it is not clearly brought out that tumors of the posterior association area or concrete memory field of the brain will necessarily cause some disturbances of mentality, especially those in the domain of orientation and in the sensorial field; (3) that tumors anywhere situated may cause mental disorders simply through the disturbance or impairment of association which must result; (4) sufficiently close attention is not paid to the particular point that the psychic disorders caused by prefrontal tumors are especially those of the higher intellectual functions, and that in many of the cases studied the normal mental status of the individual is not properly taken into account; and (5) the fact is not recognized that in tumors situated in one of the prefrontal regions, especially in the right, the individual's intellectual faculties may be largely preserved because of the integrity of the corresponding region of the other side.

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[4] Schuster, Psychische Storungen bei Hirntumoren, Stuttgart, 1902.

[5] Von Monakow, Ergebnisse der Physiologie, vol. 3, No. 2, 1904.

We can only arrive at satisfactory conclusions by the closest and most thorough clinicopathologic or clinicosurgical studies of prefrontal tumors.

The following case recently observed is one of unusual interest because of the strict limitation of the lesion to the prefrontal lobe as defined in this paper, namely, that part of the cerebral hemisphere which is situated laterally and mesally cephalad of a line drawn vertically through the pre-Sylvian fissure, and on the inferior surface of the brain is cephalad of the Sylvian fossa:

X. Y., 71 years old, was a well-known physician who had, up to within a few months of his death, pursued his profession successfully, at the same time taking a moderate part in the management of other business interests.

About two months before his death he was first seen by Dr. Mills, who had been consulted by his son regarding him some weeks previously. His symptoms at this time were all mental, being chiefly exhibited in defects of memory and judgment.

A careful examination was not made at this time, as it was the desire of his family not to have him recognize the fact that his condition was being made the subject of investigation. He had not any paralytic or sensory symptoms, however, and, so far as could be determined by the examination that was made, none in the domain of his special senses. Unfortunately, an ophthalmoscopic examination was not made. His symptoms appeared at the time to correspond to those frequently seen in cases of cerebral arteriosclerosis.

He had frequently put his hand to the left side of his head, as if suffering from uncomfortable sensations, but he did not call attention to any pain or distress in his head by speaking of it.

Shortly after this he was noticed to have a shuffling gait, and later he was unable to convey food to his mouth with any certainty. Soon after he began to have muscular tremors in his hands and legs, much like those seen in paralysis agitans. At first they were on the left side only, but toward the end involved the right side also. They were at first partially under control, and by an effort of will he could stop them; later he could not, and they occurred also during sleep.

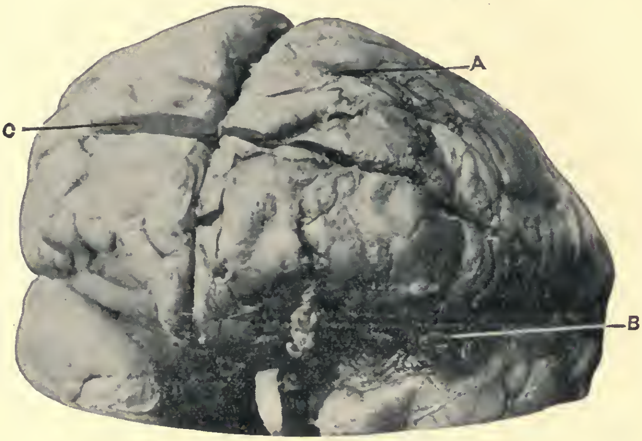

Fig. 1.—Photograph showing the tumor as viewed in front, laterally and at the base. *a*, upper limit of the tumor; *b*, recent hemorrhage; *c*, the line of the longitudinal section through the cerebrum.

About six weeks before his death he had occasional turns of vomiting for about a week; a week later one or two more. These were the only attacks of vomiting he had during the whole course of his disease.

About two weeks before his death the patient suddenly became unconscious, this condition lasting for three days. He then rallied and could be aroused during part of the day and answer questions. The comatose condition gradually returned and he died very quietly with no convulsions or any unusual symptoms.

At our request the patient's son, an unusually observant and well-equipped physician, sent us a description of his symptoms from the time when they were first noted by members of his family. The patient died April 19, 1905. Attention was not particularly directed to any evidence of mental failure until December, 1904, although his son stated that on looking back he felt sure it began much earlier. In the summer of 1903 he seemed to have lost, to some extent, his grasp as a physician, he having at this period taken charge of his son's practice during the latter's absence on a vacation.

In the summer and fall of 1904 it was observed that he was no longer attentive to the care of his house, he having formerly been particular in this respect. At the table he became more careless, as in carving and carrying food to his mouth. He was indifferent as to what occurred, as when he dropped food on the table or in his lap.

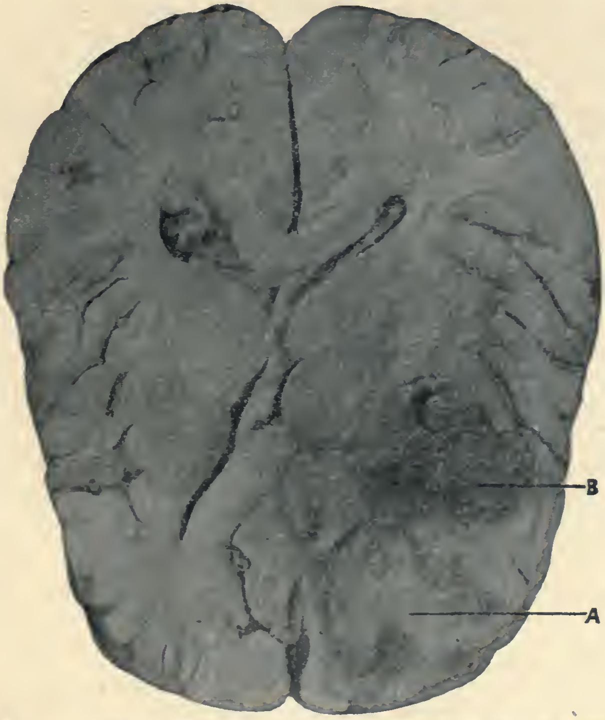

Fig. 2.—Longitudinal section of the cerebrum showing the extent of the tumor in section and also the position and extent of the recent hemorrhage posterior to the tumor; *a*, the tumor; *b*, the hemorrhage.

His loss of memory seemed to develop rather suddenly about Dec. 1, 1904. Memory for recent events failed first; he soon forgot his recent patients and would ride about hunting those he had not seen for years. He even made charges in his books to patients who had been dead for years. He never seemed to lose himself, and in his drives about the country within a month of his death he always seemed to know where he was.

A gradual loss of judgment and comparison had occurred after the fall of 1904. When, for instance, his son consulted him about some business matters which he had known all about better than any one else he answered that he did not know and that so and so had better be consulted. A slightly delusional state developed. This was mostly with reference to medical work. He had an idea that he was very busy, that he was sent for to various places, and he would hurry around to do what he thought was his work. He would stop at all sorts of places, hunting the persons who were sick. In the evening he would try to tell where he had been, but got hopelessly confused. Once he thought for a number of evenings in succession that he had theater tickets and must get ready to go, although scarcely in his life had he been to the theater. On account of his wife's deafness he was in the habit of giving her verbal reports of sermons. During the few months preceding the development of marked symptoms she noticed his accounts were becoming confused, and that he could not give a clear idea of what was said.

Another quite early symptom was change in disposition. He would sit at meals without saying anything, and became exceedingly mild and gentle. His ordinary greeting became unnaturally cordial.

No marked change occurred in writing until about December, 1904, when it became decidedly more careless. Spelling was not affected, although he frequently used a wrong word even a month or two earlier. During December his letters became confused and careless. He would start with an idea and then become bewildered, and would repeat the same words and phrases several times.

The necropsy was made about twenty-four hours after death. As the body had been carefully injected with formalin, on removing the calvarium the brain in general was found to be in an unusually good state of preservation. On attempting to remove the brain from the skull the anterior pole of the left hemisphere was found to be adherent to the skull, necessitating careful dissection of the adhesions at this point. A small quantity of blood was seen oozing from the left Sylvian fissure at its anterior inferior extremity. It was evident while the brain was still in position that an abnormal mass was present in the left prefrontal region, the brain substance, the membranes and the lesion present not being separable.

The lesion was found to be a prefrontal tumor, which was fleshy in color, hard in consistence and well-defined from the surrounding tissue. Studied from its lateral aspect, the tumor was absolutely prefrontal, occupying the anterior third of the first frontal gyre, the anterior fourth of the second and slightly less of the third. It occupied practically the entire orbital surface and on the mesal aspect the entire area cephalad and ventral of the knee of the callosum; it also involved the collosum itself, the mass projecting toward the other hemisphere. A good idea of the lateral and the anterior position of the tumor is given by the photograph (Fig. 1).

A horizontal section of the entire brain made just below the level of the callosum showed that the growth involved the entire subcortex and cortex of the first and second frontal gyres, but only the orbital portion of the cortex of the third frontal, laterally the outskirts of the growth only reaching to within three-eighths of an inch of the cortex. The tumor extended backward so as to involve the anterior extremities of the caudatum and internal capsule, as shown in the photograph (Fig. 2).

Caudad of the tumor in the Sylvian fissure, a fresh hemorrhage was found, this having destroyed in large part the insula. The nerve cells of the cortex, as shown by the Betz cells in the paracentral lobule, were in a fair state of preservation. Here and there was found a diseased cell, but these were not numerous. In these cells the nuclei were found in the periphery and the chromophylic elements stained diffusely. The pia was of normal thickness. The arteries throughout the entire brain were markedly sclerotic; in fact, they were so much so that at the base of the brain the anterior cerebral arteries pressed upon the optic chiasm, causing the optic nerve on each side to assume a semi-lunar shape. On cross-section the optic nerves showed a slight amount of degeneration. Sections taken from different parts of the cortex and from the oblongata did not show any alterations.

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SEPARATE SENSORY CENTERS IN THE PARIETAL LOBE FOR THE LIMBS.[1]

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Friedrich Müller has recently made a careful study of the disturbances of sensation resulting from lesions of the brain. He mentions that cerebral hemianesthesia without motor paralysis is seldom seen, if hysterical hemianesthesia be excluded, and that many authors deny the possibility of such an occurrence. Dejerine, among others, has said that cerebral hemianesthesia is always associated with hemiplegia, and that the most intense sensory alterations are found in the weaker limb, although no parallelism exists between the degree of the hemiplegia and that of the hemianesthesia.

Munk, Tripier, Dejerine, Henschen and others hold that the motor cortical areas are also sensory, and Dejerine says he has never seen a lesion in the region of the supramarginal gyrus which caused sensory disturbances without motor disturbances. One must acknowledge, according to Müller, that cortical sensory disturbances seldom occur without weakness of the limbs in which sensation is disturbed.

Von Monakow has accepted the view that the motor area is confined to the precentral convolution, and that the sensory region is in the postcentral convolution and parietal lobe; and he refers in support of this view to cases of cortical hemiplegia without hemianesthesia and to cases of cortical hemianesthesia without hemiplegia. The experience of surgeons seems to show that the precentral convolution is not an essential part of the sensory area, although it cannot be denied that a lesion of this convolution may occasionally cause disturbance of stereognosis. Von Monakow holds, from a study of secondary degeneration, that the pyramidal tract arises only in the precentral convolution.

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[1] Read before the Philadelphia Neurological Society, Dec. 19. 1905. From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania, and from the Polyclinic Hospital.

This is the presentation of the subject as given by Müller, and to the cases of hemianesthesia without hemiplegia from lesions of the parietal lobe mentioned by v. Monakow, (cases of Cox, Mills, Redlich, Spiller, Oppenheim, Durante, v. Monakow), Müller adds the cases of Prevost, Wilbrand, Lang, Henschen (4), Jany, Long, and five cases of his own. He shows that subjective disturbances of sensation (paresthesia) may be more extensive than objective sensory disturbances from a cortical lesion.[2]

Among American writers the one who has longest and most persistently maintained the separation of the motor and sensory areas of the brain is C. K. Mills. At the meeting of the American Neurological Association in St. Louis in Sept. 1904, he suggested that the cortical sensory area for the upper limb is probably separate from that for the lower limb. This view has been entertained by others also, thus Alfred W. Campbell[3] says: "I incline to the belief that the area for common sensation, like the motor area, will ultimately be divided into different levels or centers." Doubtless many others have held a similar opinion, but there has been little real foundation for it. In the same paper Campbell remarks: "Proof, from the pathological side, of the correctness of this localisation [of the motor area anterior to the central fissure] has been supplied by an examination of the brains of cases of amyotrophic lateral sclerosis. Amyotrophic lateral sclerosis is a perfect example of a malady limited in its affection to the muscles and the motor system of neurones; and on making serial sections of the central gyri in two typical instances, it was extremely instructive to find profound changes confined absolutely to our 'precentral or motor area' and consisting essentially of a destruction of the cells of Betz."

The idea of employing cases of amyotrophic lateral sclerosis to limit the motor area, was suggested by me[4] in 1900, and in my report of a case of this disease studied with this object of localization in view by the Marchi method, mention is made of degenerated fibers in the postcentral convolution. They were fewer by far than were the degenerated fibers in the precentral convolution, but they were unquestionably present. I have recently re-examined these sections. It is true that the relative intensity of the degeneration in the postcentral and precentral convolutions, as represented by the artist in the diagrammatic drawing accompanying my paper, is not correct. The difference in the results reached by Campbell and by me may depend on the fact that he relied on alteration of the cells of Betz, and I on degeneration of nerve fibers as shown by the Marchi method.

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[2] Friedrich Müller, Sammlung Klinischer Vorträge, Innere Medizin, No. 118-19, 1905.

[3] Campbell. The Journal of Mental Science, Jan. 1904, p. 651.

I have had recently an opportunity to study a case which shows that sensation may be affected from a cerebral cortical lesion, without motor paralysis; but still more important, that the sensory alteration may be confined to one limb. From this we may conclude that the sensory centers for the limbs are distinct from one another.

M. W., male, 38 years of age, teacher, came to my clinic at the Polyclinic Hospital, in Aug. or Sept., 1905.

He was perfectly well until July 21, 1904. He was struck on that day over the right parietal lobe with a club by a man who intended to injure him. He was unconscious one or two hours and was confined to his bed about ten days after he received the blow. He was unable to move the fingers of his left hand during the two weeks following the injury and could not make a fist. It is impossible to say whether this inability was the result of motor weakness or of sensory disturbances. He could move his lower limb the day following the injury. He is uncertain as to whether the lower part of his face was paretic or not. The hand at first felt "dead," and he has had paresthesia in it since the injury. He has never had any subjective or objective disturbances of sensation in the left lower limb or face.

Present condition, Dec. 11, 1905: The movements of the left upper limb are awkward, although the limb can be moved freely at all parts, and is not weak. He is unable to button his coat with his left hand alone when his eyes are closed, because of incoordination of the fingers, but can button it promptly with his right hand. The sense of position is greatly impaired in the fingers of his left hand, and he is usually unable to tell which finger is moved, or when a finger is bent backward or forward. Stereognostic perception is also greatly impaired in his left hand, and usually he is unable to name any object placed in this hand if he does not see it. Occasionally if the object falls from the hand he is unaware of the loss. Sensations of touch, pain and temperature are diminished in the left hand, but not to the same degree as are the sense of position and stereognostic perception. He will occasionally answer correctly when his left hand is touched or pricked with a pin, but usually his answers are uncertain and in marked contrast to the positiveness with which he replies to sensory tests of the right hand. The sensation of touch is a little less acute in the left hand than in the left arm above the elbow. Pain sensation is less altered than tactile sensation, and temperature sensations for both cold and warmth are more nearly equal in the two upper extremities. The spacing sensation is also greatly altered in the left hand, and he cannot tell whether the thumb and first finger are near together or far apart. In the right hand he can tell whether the thumb and first finger are one or two inches apart. Sensations on the chest seem to be the same on the two sides.

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[4] Spiller. THE JOURNAL OF NERVOUS AND MENTAL DISEASE, 1900, p. 165, and Contributions from the Wm. Pepper Laboratory of Clinical Medicine, vol. 1, 1900.

The left lower limb is not affected. His answers to sensory stimuli are always correct and prompt. He recognizes at once any movement of the toes and the direction in which they are moved. The limb is not weak. The patellar reflexes are not prompt. Sensation in the left side of the face appears to be entirely normal, and the right side of the body is not affected. Hemianopsia is not present.

Extremely interesting in this case are the marked sensory alterations of the left upper limb, with no disturbances of the left side of the face and of the left lower limb, and no motor palsy of the left limbs, at least at the present time; the symptoms resulting from a blow given over the right parietal lobe. The case seems to indicate that the sensory center for the upper limb must be distinct from those for the face and lower limb. I am inclined to place the lesion in the lower part of the parietal lobe, in part because this area is near the motor center for the upper limb, and in part because of a case I[5] reported in 1898. This was as follows: A man complained, during the night preceding his attack, of fatigue, headache and inability to sleep. The following morning he fell to the floor on attempting to arise from his bed, but was not unconscious. When he was found he had paresis of the left limbs and of the left side of the face and tongue. Later the patient could raise his left upper limb above his head, but every movement of this limb was ataxic in an extreme degree, though the limb was not paralyzed. The mental condition of the patient prevented an examination of the condition of sensation. At the necropsy a hemorrhage was found in the right parietal lobe. The hemorrhagic area was about 2 centimetres in diameter, and extended inward in the form of a cone to the lateral ventricle, having its base in the cortex. It was situated about 4 centimetres from the longitudinal fissure, and about 2 centimetres or 3 centimetres behind the Rolandic fissure. The injury was in, or very close to, the supramarginal gyrus. The ataxia was probably caused by a loss of the sense of position.

The case that I now report seems to indicate that the alterations of the sensations of position and space and of the stereognostic perception is greater from a lesion of the parietal lobe than is the alteration of the sensations of touch, pain and temperature.

In a paper published in 1904 I[6] discussed the location of certain forms of sensation in the parietal lobe, and reported two cases with necropsy. In one of these the loss of stereognostic perception and of the sense of position in the left hand, the awkward movements of the left fingers, and the impairment of sensation for touch, but not pain, in the left upper limb were attributable to tuberculous plaques almost confined to the right parietal lobe. I could find no case of this tuberculous meningitis in plaques described in American literature, and few in foreign. The other case was one in which ataxia, paresis and astereognosis of the left upper limb were caused by softening of the right parietal lobe.

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[5] Spiller. JOURNAL OF NERVOUS AND MENTAL DISEASE, Jan., 1899, p. 43.

[6] Spiller. "A Report of Five Cases of Tumor of the Brain with Necropsy." The American Journal of the Medical Sciences, Feb., 1904, p. 311.

Extracted from the American Journal of the Medical Sciences, March, 1906

TREATMENT OF SELECTED CASES OF CEREBRAL, SPINAL, AND PERIPHERAL NERVE PALSIES AND ATHETOSIS BY NERVE TRANSPLANTATION.[1]

WITH THE REPORT OF A CASE OF ATHETOSIS BENEFITED BY OPERATION.

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REMARKS BY DR. SPILLER.

A new field seems to have been opened in the treatment of selected cases of paralysis, whether they are of cerebral, spinal, or peripheral nerve origin, and also in the treatment of athetosis. It is not too much to say that the results obtained in cases of acute anterior poliomyelitis by nerve transplantation have been so encouraging that the operation for recovery from the paralysis in selected cases of this disorder may now be regarded as passing beyond the experimental stage.

Acute anterior poliomyelitis is a disease of a more or less limited number of nerve cells in the anterior horn or horns of the spinal cord. In 1902 it occurred to me that we should be able by nerve transplantation in acute anterior poliomyelitis to bring diseased peripheral nerve fibres into union with healthy nerves and thereby to restore function. Such a procedure would be more advantageous than a tendon transplantation, as by the former method the paralyzed muscles would be restored if the operation were successful. The subject has been discussed in a paper by Dr. Frazier and myself,[1] read before the American Medical Association.

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[1] The paper was read and the patient was exhibited before the College of Physicians November 1, 1905.

In 1902 I suggested to Dr. C. H. Frazier and Dr. J. K. Young that we might with propriety try nerve transplantation in cases of acute anterior poliomyelitis where the palsy was not very extensive. The operation was performed on December 4, 1902, by Dr. Young, and the patient was exhibited by Dr. Young and myself before the Philadelphia Neurological Society February 24, 1903, and the case was reported in the *Journal of Nervous and Mental Disease,* in the proceedings of the Society, June, 1903. Even in February, 1903, some return of power could be observed, and at the present time the improvement is distinct. The case has been reported by Dr. Young before the American Orthopædic Association June, 1904, and also in a paper by Dr. Frazier and myself,[2] together with the report of another case in which the operation had been done by Dr. Frazier almost at the same time.

The chief dangers in operating on cases of anterior poliomyelitis are delayed union and overgrowth of connective tissue in the nerve at the site of operation. Where a whole nerve is diseased and is so large as the peroneal, it is probably necessary to cut a portion of the sound nerve used for grafting, either transversely or transversely and longitudinally, and when the latter is done to insert the split portion into the diseased nerve; but where the fibres are small, as are those supplying the anterior tibial muscle, probably a sufficient number of nerve fibres are cut in the healthy nerve by the longitudinal splitting and the insertion of the diseased fibres within the slit. The new axis cylinders which develop after an injury of nerve fibres are more numerous than those existing before the operation, and probably by growing into the diseased nerve, if we accept this view of regeneration, suffice for its innervation. If we accept the peripheral theory of regeneration, the axis cylinders that develop in the peripheral end of the nerve are sufficient for the restoration of function. It hardly seems advisable to cut a healthy nerve entirely across, as Kilvington suggests, because if an excessive amount of connective tissue forms reunion with functional restoration of the formerly healthy fibres may not occur.

Peckham,[3] as I found later, cut two of the four branches passing from the internal popliteal nerve into the heads of the gastrocnemius in a case of anterior poliomyelitis of ten years' duration and inserted them into the diseased nerve. At the end of six weeks the toes could be extended (dorsal flexion) until they were perfectly straight, a motion that had been impossible until after this operation. There was no apparent weakness in the gastrocnemius and soleus muscles. The same operation was performed in another case for paralysis resulting from fracture of the spine. The results were not very gratifying in either case, and his paper seems to have received little or no attention.

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[1] The Journal of the American Medical Association, January 21, 1905.

[2] Loc. cit.

[3] The Providence Medical Journal, January, 1900, p. 5.

The paralysis in cases of acute anterior poliomyelitis is often confined to a small group of muscles, as shown in a recent paper by Walther Baumann.[1] Baumann examined 85 cases of acute anterior poliomyelitis. Complete paralysis of one or both lower limbs occurred in 16 cases; complete paralysis of the lower limbs, except that the movement of the toes was preserved, in 5 cases; complete paralysis of the lower limbs, except that the movements of the extensor digitorum communis and the peronei were preserved, in 1 case.

Paralysis of only the	dorsal flexors of the foot	occurred in	29 cases.
" "	extensors of the knee	"	12 "
" "	peronei	"	10 "
" "	calf muscles	"	8 "
" "	flexors of the knee	"	8 "
" "	tibialis anticus	"	7 "
" "	glutei	"	6 "
" "	adductors	"	2 "

Complete paralysis of one or both upper limbs occurred in 3 cases; complete paralysis, except that the movements of the fingers were preserved, in 2 cases.

Paralysis of only the	deltoideus	occurred in	8 cases.
" "	flexors of the forearm	"	5 "
" "	thenar eminence	"	5 "
" "	pectoralis major and supinator longus	"	3 "
" "	triceps	"	4 "
" "	small muscles of the hand	"	2 "
" "	supraspinatus	"	2 "
" "	entire musculospiral territory	"	1 "

From this table it will readily be seen that partial paralysis is very common in anterior poliomyelitis, and especially noteworthy is the large number of cases, 29, in which the dorsal flexors of the foot were paralyzed. It is probably because of this that attention has been directed chiefly to the lower limbs as regards nerve transplantation in acute anterior poliomyelitis. The paralysis of the upper limbs, however, is amenable to treatment by nerve transplantation.

Hackenbruch[1] reported at the meeting of the Kongress der Deutschen Gesellschaft für Chirurgie, June, 1903, and therefore after the presentation of the case by Dr. Young and myself in

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[1] Monatsschrift f. Psychiatrie und Neurologie, June, 1905, p. 485.

[2] Archiv f. klin. Chirurgie, 1903, No. 3, Bd. lxxi. p. 633; also Deutsche med. Wochenschrift, June 22, 1905, No. 25, p. 986.

February, 1903, that he had inserted a third of the tibial nerve into the paralyzed peroneal nerve in a case of poliomyelitis. In June, 1905, he reported the results obtained in this case and in two others. Two cases were only partially successful, but the third gave most gratifying results.

This third case was one of peroneal palsy, and the palsy seems to have been complete in the distribution of the peroneal nerve. A third of the posterior tibial nerve was inserted into the peroneal. Slight improvement was seen four months after the operation, but a year and a half after the operation the movements of the lower limb and foot were good, the patient was able to elevate herself upon the toes of this foot, and to stand on this foot alone. The limb had become 1 cm. longer and the foot had become larger. The limb, as a whole, was a little weaker than the other.

Many experiments have been performed in nerve transplantation on animals since the first operation by Flourens. Basil Kilvington[1] gives a very good presentation of this subject, and then makes an application to surgery. He suggests that nerve transplantation might be used in cases of anterior poliomyelitis, although he acknowledges that he has not performed the operation on the human subject. As he makes no reference to the work of others in so far as it relates to man, he was probably ignorant of the results already accomplished in cases of anterior poliomyelitis. His suggestion is to suture the nerve supplying the paralyzed muscles on to the cut surface of the proximal end of an adjacent healthy nerve. The healthy nerve may be cut partly across and the paralyzed nerve fixed in the gap so formed, or the healthy nerve may be cut completely across and its own peripheral end, as well as the peripheral end of the paralyzed nerve, may be sutured to the central end. This hardly seems a wise proceeding to me. Kilvington published his paper April 29, 1905.

Harris[2] and Low reported before the Clinical Society of London, October, 1904, a case of infantile paralysis in which they had done nerve suture in order to overcome paralysis of the right deltoid and spinati muscles. The operation is described by Harris in an abstract as follows (the original paper I have not as yet been able to obtain[3]): The fifth cervical nerve was split in halves, and the upper bundle, which caused no muscular contraction on faradic stimulation, save fibrillary movements in the deltoid, was cut across as near the foramen as possible, and turned down in front of the remaining half of the fifth nerve and then sutured into a nick cut in the front of the sixth cervical nerve. The uncut portion of the fifth nerve contained the fibres for the biceps, as faradic stimulation of this bundle caused powerful flexion of the forearm. In this way the degenerated bundle in the fifth nerve, supplying the deltoid, was sutured to a healthy nerve so as to allow regeneration to take place by the formation of a connection with the healthy fibres in the sixth nerve. For sixth months after the operation no sign of improvement appeared, in spite of regular massage and galvanism. Then, eleven months after the onset of the paralysis, slight improvement in the power of the shoulder was noticed, and the progress, at first slow, afterward more rapid, was maintained, so that ultimately the child could put up her arm readily straight above her head.

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[1] British Medical Journal, April 29, 1905, p. 935.

[2] The Practitioner, August, 1905, p. 228.

[3] I have, since our paper was read, obtained the original article by Harris and Low.

It is therefore true, I think, that the operation of nerve transplantation for acute anterior poliomyelitis where the paralysis is not too extensive is passing beyond the experimental stage. The application of nerve transplantation to peripheral nerve palsies is apparent.

In most cases of hemiplegia the paralysis is greater in the extensors of the upper limb and in the flexors of the lower limb, but, as a rule, the difference is not sufficiently marked to justify the operation of nerve transplantation. Occasionally, however, the palsy is much more intense in certain muscles. Fischer[1] recently, in speaking of this subject, states that the weakness of the extensors of the fingers in rare cases of cerebral lesion may be so pronounced that the paralysis may have the appearance of musculospiral palsy, as in the cases of Pick and Oppenheim. I have observed a distinct difference in the degree of paralysis in the extensor and flexor groups of muscles in a number of cases in which hemiplegia had developed early in life.

It has been my endeavor for several years to devise some method of treatment for athetosis. This condition is observed only when some return of function has occurred, or at least when the paralysis is incomplete, and if the movements are violent the muscles hypertrophy because of their constant use; the patient with severe athetosis, however, has little or no voluntary control of his limbs, and the flexors are usually much more powerful than the extensors in the upper limbs. It occurred to me that if we could switch off, so to speak, some of this excessive innervation of the flexors into the extensors by nerve transplantation, we might be able to establish a more nearly normal relation between certain groups of muscles and their opponents, and by division of nerves be able to lessen the athetoid movements probably permanently. It hardly seemed likely that restoration of function would be so perfect as to permit a return of athetoid spasms. This subject was presented by Dr. Frazier and myself[2] in a paper read before the New York Neurological Society, March 7, 1905. At that meeting Dr. G. L. Walton remarked that in a case coming under his observation the athetosis was so troublesome that the arm was amputated at the shoulder at the request of the patient.

A case of extremely violent athetosis has been under my care about five or six years at irregular periods. The young man had probably had the movements all his life, except possibly during his first year, and had no control over his powerful muscles. His mother had been obliged for years to dress him with the greatest difficulty every morning. His upper limbs were jerked about most violently, but the flexor muscles were far more powerful than the extensors. The case seemed most suitable for an attempt at nerve transplantation, and was referred by me to Dr. Frazier for this purpose. The method of operation will be described by Dr. Frazier.

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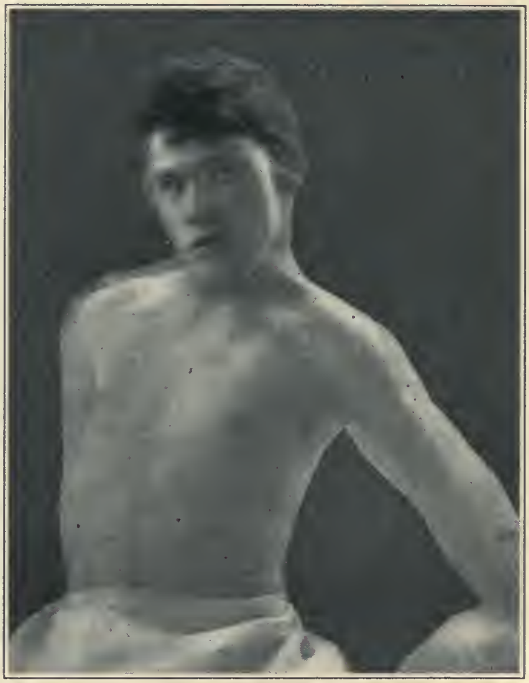
[1] Monatsschrift f. Psychiatrie und Neurologie, August, 1905, p. 98.

[2] Journal of Nervous and Mental Disease, May, 1905, p. 310; Walton, ibid., October, 1905, p. 655.

FIG. 1

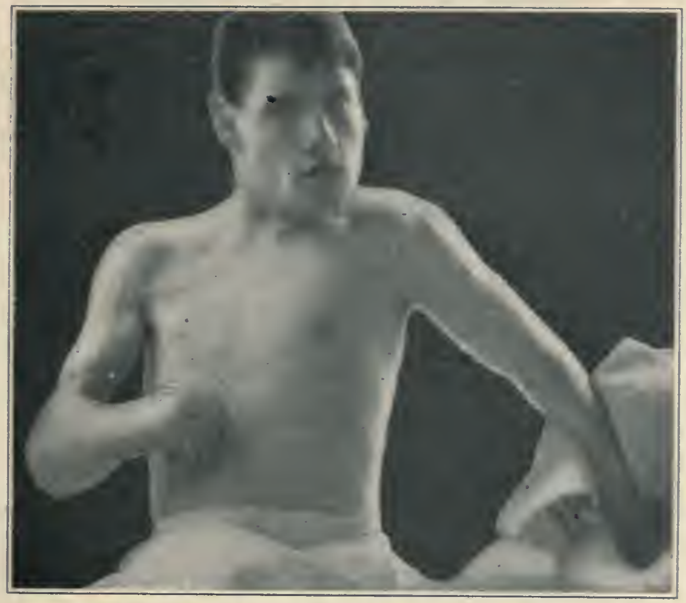


Photograph taken before the first operation, indistinct despite the fact that it was instantaneous. It shows how constant and uncontrollable were the athetoid movements and shows also the intensity of the spasms. Much of the blurring of outlines has been removed in the reproduction.

The case was presented at the meeting of the American Neurological Association, June 2, 1905, two months and a half after the first operation, and at that time there was distinct return of power at the wrist, in the flexors of the fingers, and at the elbow, but the movements were by no means normal. The presentation was made at that time in order that the many neurologists present might be able to judge for themselves of the degree of athetosis that had existed before the operation, inasmuch as the right upper limb had not been operated upon at the time of the presentation; and in order that the developments of the case, whatever they might be, could be watched. We made no claims—nor do we now—but presented the results of a hitherto unattempted operation for the relief of a most distressing condition, believing that, whether failure or success were the ultimate outcome, the results should be made known.

As early as two and a half months after the operation the condition of the patient was so much improved that he was well satisfied with the results, and stated he had never in his life been so comfortable. We had established at that time that it was possible to remove the athetosis if we caused a partial paralysis, and we could argue that should the athetosis return it would do so only with the disappearance of the paralysis. In case of return of athetosis we should be able to diminish the voluntary power, and thereby also the athetosis, by some other operation.

FIG. 2



Instantaneous photograph taken before the first operation, showing the violence of the spasms.

It may be said that the operation was an attempt to influence a cerebral lesion by disturbing peripheral nerves, and this indeed is what it was. We could not remove the source of the irritation, but if we could remove the manifestations of this irritation without causing the patient more discomfort, we might consider the operation a success. This is what we have accomplished, at least in part. There is no doubt that this man's condition is far better than it was before any operation was attempted, and we may hope for a still greater restoration of power. He has now returned to his occupation of selling papers and is very happy over his improved condition.

Massage and electricity have done much to keep up the tone of the muscles, and the patient has made frequent attempts to employ his upper limbs. He is now being treated by systematic gymnastic exercises.

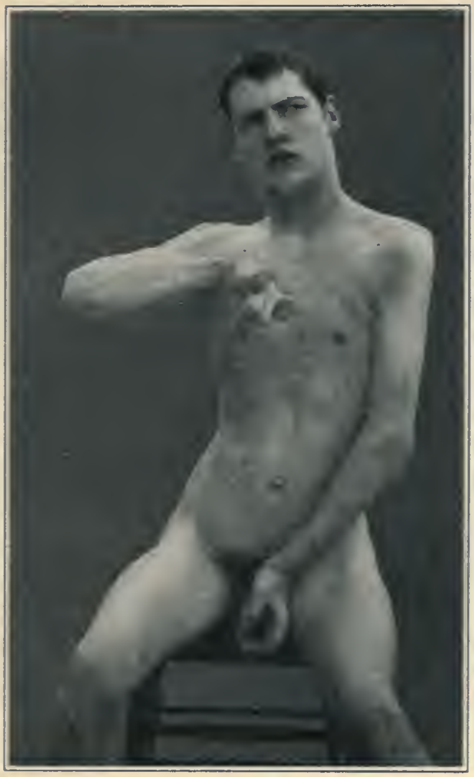
Seven and a half months have passed since the first operation, and the results are most encouraging and lead us to believe that athetosis will not return, or if it should, that it will be far less severe than before the first operation. It is important to have established that with partial paralysis, produced by operation on nerves, athetosis disappears. No one, I think, who was familiar with this man's condition before any operation was performed, and sees him now, would dispute that he is far more comfortable than at any previous period of his life.

If athetosis is the result of irritation of central motor fibres it seems strange that no symptoms are produced by the prevention of any manifestation of this irritation. One might suppose that the impulses sent over the motor tracts, having been arrested, would flow over into fibres destined for the innervation of the lower limbs, or that some evidence of discomfort would be observed. Nothing of the kind occurred. It is said that sometimes in epilepsy, when the convulsion is arrested, the patient's condition is worse than if the convulsive seizure had freed the stored-up energy, and we might expect a somewhat similar result when the irritation of athetosis finds no outward expression, but such does not seem to be the case.

Dr. Purves Stewart recently suggested to me, after seeing the patient, that if in epilepsy we could cut all the motor nerves we should arrest the convulsions and produce a condition similar to that seen in our patient so far as arrest of involuntary movements is concerned, but we should not cure the epilepsy. I fully agree with him in this statement, but if we have arrested the athetoid movements, even though we have not removed the cerebral lesion, we have accomplished much. Epilepsy is more than convulsions, but if it were a practical operation to cut the nerves for the prevention of spasm, which of course it is not, we might resort to it.

The operation performed in this case of athetosis shows convincingly that when the peripheral end of a healthy nerve is inserted into another healthy nerve by a longitudinal and not transverse incision in the latter, return of function may occur in the inserted nerve. The musculospiral nerve was not cut transversely, and yet partial return of function has occurred in the distributions of the median and ulnar nerves. The results justify the end-to-side anastomosis—the peripheral end of the affected nerve inserted into the unaffected nerve—we have advised for the treatment of the paralysis of anterior poliomyelitis confined to one or two muscles. There is unquestionably a difference between using the peripheral end of a healthy nerve and the peripheral end of a diseased nerve, but if the nerve is much affected it is not likely to regenerate by any method of transplantation, and it is because of this that the prognosis is better the earlier the operation is done. This method of end-to-side anastomosis, however, would probably not be advisable in those cases in which the entire nerve distribution such as the peroneal, were affected, and it would probably be necessary, under such circumstances, to insert some healthy fibres into the diseased nerve.

FIG. 3

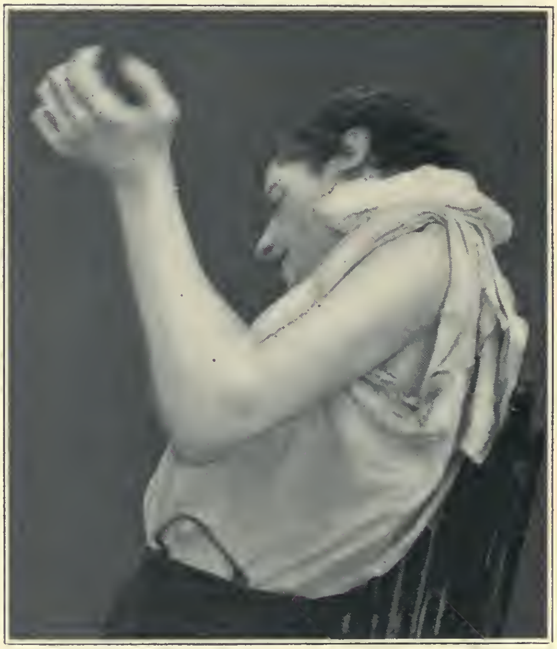


Photograph taken soon after the first operation, showing the immediate paralysis of the left upper limb and marked athetoid movements of the right.

The experimental operations noted below were performed at the suggestion of Drs. Spiller and Frazier under the direction of Dr. Van Kaathoven. The two cases cited are chosen as representative of a series of similar experiments giving similar results. This experimental work was done partly to demonstrate certain questions regarding restoration of function and the operative technique, and was performed in connection with the voluntary course in operative surgery.

CASE I.—Mongrel dog. Operation. An incision was made on the anterior aspect of the thigh over the femoral vessels and the anterior crural nerve exposed and freed. A posterior incision half way between the great trochanter and tuberosity of the ischium was then made, the muscles separated, and the sciatic nerve exposed and freed. An opening was then made through the adductor magnus, this perforation allowing of communication between the anterior and posterior aspects of the thigh. The two nerves were then cut. By means of silk sutures through the sheath the peripheral portion of the anterior crural was sutured to the proximal part of the sciatic, and the peripheral sciatic to the proximal anterior crural. The leg was then partially immobilized, the superficial wounds having been closed by means of silkworm sutures. Result: six days after operation the wounds were examined and found to have united by first intention. Stitches and bandage were removed. During the middle of the second week the dog commenced to move the injured leg. At the beginning of the third week restoration of

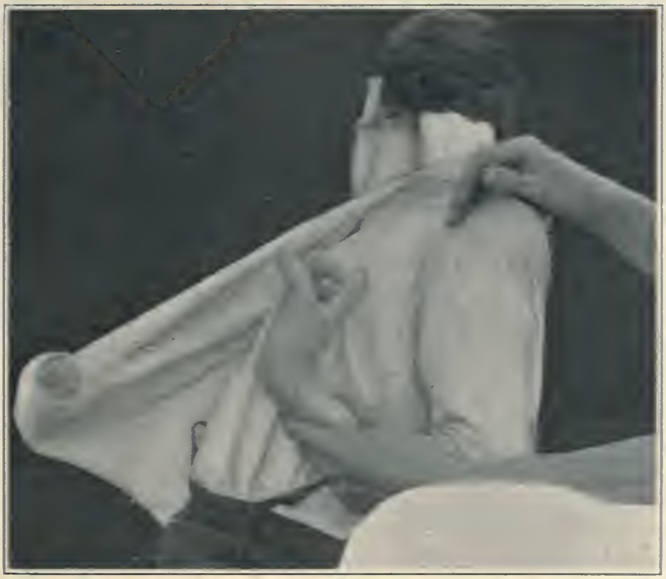
FIG. 4



Photograph taken July 14, 1905, 120 days after the first operation, showing flexion of the forearm on arm as an evidence of gradual restoration of voluntary movements.

function became more marked. It was observed that when walking the animal extended its hind leg instead of flexing it. After three or four unsuccessful attempts at walking on the injured foot the animal apparently became discouraged and let the leg hang down, scampering away on three legs. About the onset of the fourth week the dog had apparently learned to control the movements of the hind leg, although when running he still frequently extended the leg instead of flexing it. During the latter part of the week the animal had improved markedly in its locomotion, the only defect being a turning under of the foot, apparently due to inability to extend it freely. At the end of the fifth week he showed marked improvement in this condition, now only turning the foot under occasionally, apparently only when running or excited. During the sixth and seventh weeks the animal practically walked as well as before section of the nerves. The results of this experiment were exactly similar to those of earlier ones, the same operation having been performed.

FIG. 5



Photograph taken July 14, 1905, 120 days after the first operation, showing flexion of the hand on the forearm as an evidence of return of voluntary movements.

CASE II.—Terrier. A similar operation was performed upon the brachial plexus. The nerves being reached by a transverse section of the pectoralis major and a separation of the fibres of the pectoralis minor. The median and musculospiral nerves were chosen for the experiment. Suturing was done as before—peripheral median to proximal musculospiral and proximal median to peripheral musculospiral. Aside from some superficial suppuration the dog made a good operative recovery. No restoration of function followed operation for eight weeks. There was some contracture at the elbow joint, none at the wrist. After fourteen to sixteen weeks there was less contracture and less atrophy, still the dog seemed to make little or no use of its leg. The explanation of failure in the cases of experimentation on the brachial plexus lies probably in the fact that the undivided nerves, the ulnar and musculocutaneous, cause spasm of the muscle groups governed by them, as the groups innervated by the median and musculospiral are temporarily paralyzed.

HISTORY OF A CASE OF ATHETOSIS.

Frederick H., aged nineteen years, was admitted to the University Hospital on the 10th of March, 1905.

Social History. Single, native of Philadelphia. More than an average consumer of tea and coffee. Occupation, sells newspapers.

Previous Medical History. He was said to have been born at the seventh month. Had pertussis in childhood. For the past year has complained of rheumatic pains in the right leg, which have gradually grown worse. Apart from this he has always enjoyed good health.

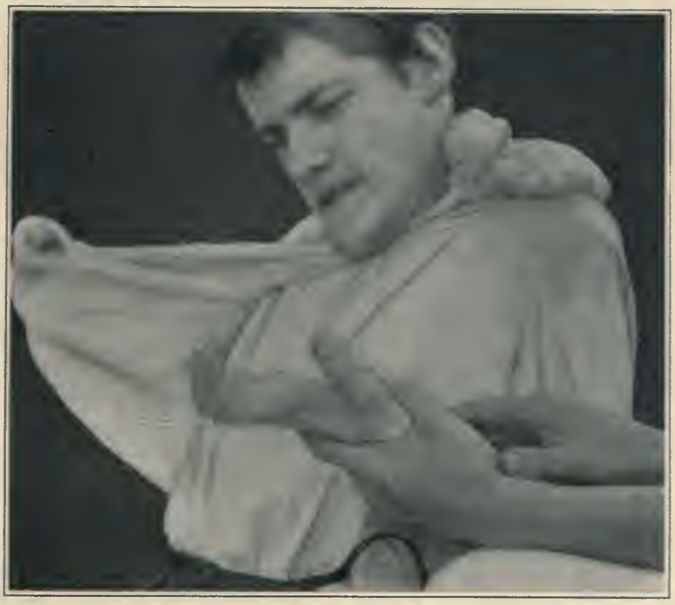
Family History. Father and mother both complain of rheumatism; the former is believed to have some disease of the kidneys. Three brothers and sisters are living and in good health. There is no tuberculous, cardiac, neoplastic, or neurotic history in the ancestry.

History of Present Condition. The patient says that when he was eight months old he fell from a hammock, alighting on his back, and that he was unconscious for two hours. Nothing, however, abnormal was noticed about the patient until he was two years old, when he was taken to the Jefferson Hospital because of the beginning athetoid movements of his hands and arms, and also because of his inability to walk. His mother was told that nothing could be done, and that his condition was not the result of the accident. From the ages between two and twelve his movements became more exaggerated and there seemed to be some disturbance in speech. He did not learn to walk until he was twelve years of age. He then slowly improved in gait until the present time. Owing to weakness in his ankles and knees and the athetoid movements of his upper extremities he frequently fell down. He had been under the care of Dr. Spiller for several years at the Polyclinic and University Hospitals and later during a part of his stay at the Philadelphia General Hospital.

While in the surgical wards of the Philadelphia General Hospital a craniotomy was performed and the cortical centre for his left upper extremity was excised. During his convalescence from this operation he had two or three convulsions, in which he lost consciousness at times for ten minutes. Since the operation his left arm has been more rigid, the pain in his right arm has been more pronounced, and his condition has grown steadily worse. The athetoid movements are very much more marked during excitement and cease entirely during sleep.

Physical Examination. Urine analysis: acid reaction; specific gravity, 1032; no albumin, no sugar; small amount of mucoid precipitate. Microscopic examination negative.

FIG. 6



Photograph taken July 14, 1905, 120 days after the first operation, showing the partial flexion of fingers as evidence of gradual restoration of voluntary movements.

Athetoid movements are not observed in the face and the cranial nerves are not affected.

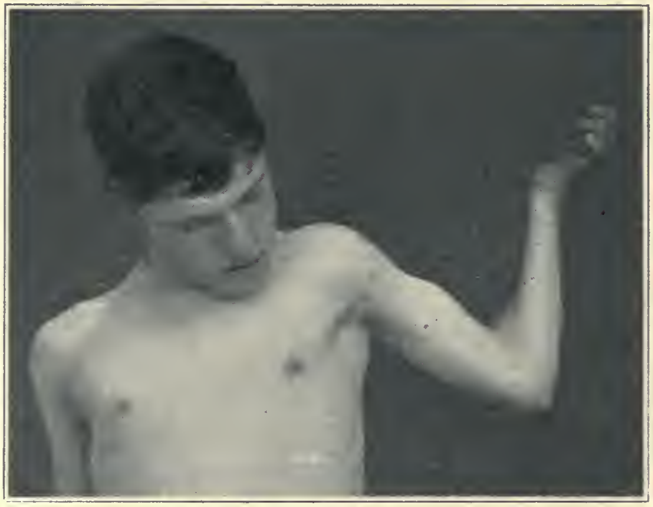
The upper limbs are in constant violent spasm and present intense athetoid movements in all parts. The flexor spasms predominate over the extensor spasms, and the hands are flexed forcibly on the forearms, and the forearms on the arms. From time to time either limb is drawn upward and backward at the shoulder, and sometimes the entire limb is drawn behind the trunk and held in this position a minute or two. In removing the coat of the patient great difficulty is experienced because of the violent spasms. The muscles of the upper limbs are unusually well developed as a result of the spastic movements, and yet the patient has not the slightest control over the upper limbs. Any attempt on his part to perform a voluntary movement with his upper limbs increases the athetosis in an extreme degree.

The lower limbs are somewhat spastic, but not sufficiently so to prevent walking. His gait is awkward and stiff because of the marked tonicity of the muscles, but athetoid movements are not observed in these limbs.

March 16, 1905. First operation on the left extremity—lateral anastomosis of the divided median and ulnar nerves to the musculospiral nerve.

17*th*. Patient complains of pain, the result of involuntary contraction of triceps and coracobrachialis. The patient can extend the fingers and the hand on the wrist.

FIG. 7



Photograph taken in December, 1905, showing the voluntary power in raising the left upper limb at the shoulder and in flexing the left forearm on the arm.

18*th*. Wound dressed and rubber tissue drain removed. Plaster-of-Paris dressing reapplied. Convulsive movements of the left arm are not so pronounced as before the operation. There is no evidence of restoration of sensation in the distribution of the median nerve.

21*st*. Patient rests quietly the greater part of the time, and is kept in bed because that position is conducive to quietude. The wound is running an aseptic course.

31*st*. Involuntary movements are chiefly, if not entirely, in the muscles of the shoulder, especially in the deltoid and biceps; sensations for touch and pain are present over the front and back of forearm, but not so acute as in the corresponding parts of the right arm. There is some involuntary movement of the triceps and at the left wrist. Sensations for touch and pain in the palmar surface of the hand are lost. They are present in the dorsal aspect of the hand, but only to a limited degree.

April 8th. Patient feels greatly benefited by the operation. It is probable that a subsequent operation will be performed, directed toward controlling the athetoid movements of the shoulder muscles.

17th. Second operation on left upper extremity performed by Dr. Van Kaathoven. The circumflex and musculocutaneous nerves were divided and an end-to-end anastomosis effected between the central end of the one and the distal end of the other, and *vice versa.*

22d. The patient has been very much more comfortable since the last operation. The muscles of the shoulders do not contract as violently as before, but there still seems to be a marked tendency toward extension in the forearm.

24th. There is some involuntary jerking on the radial side of the forearm, but this movement is slight and is in the supinator longus muscle. Involuntary movements around the shoulder have ceased. The patient seems to have no sensation for touch or pain in the palm of the hand, although it is present to some extent on the dorsal surface of the hand.

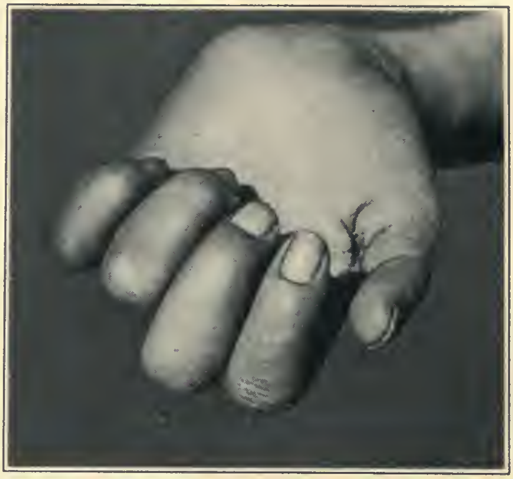
May 5th. There is very little jerking of the left upper limb, and what involuntary movement is seen is chiefly in the supinator longus muscle. The whole limb is somewhat spastic. There is considerable voluntary movement at the wrist and he can flex and extend the hand, especially when he is not excited. He can partially flex all the fingers, and the flexion is greater in the fingers nearer the ulnar side. Attempts at voluntary movements of the left hand increase greatly the athetosis of the right upper limb. The muscles of the left hand are somewhat wasted. Sensation has not returned at all in the palm of the left hand. Sensations of touch and pain are present in the left forearm anterior aspect, but diminished in the posterior part of the left forearm and left hand. Some sensation is felt in the dorsal aspect of the ulnar distribution of the hand, but none on the palmar surface of this nerve supply.

Dr. C. S. Potts reported that on this date he could detect no reaction in the muscles supplied by the ulnar and median nerves with the faradic current.

July 10th. Left upper limb. Tactile sensation is lost in the ulnar side of the palm and is felt slightly in the median side of the palm and on the anterior surface of the forearm. It is prompt in the posterior portion of the forearm and is impaired in the outer, posterior, and inner portions of the arm. Motion: the man has partial flexion of the fingers and more in the little and ring fingers; slight motion in the thumb. These movements of flexion of the fingers can be made without first extending the fingers. He has slight flexion of the wrist, more on the ulnar side (action of the flexor carpi ulnaris).

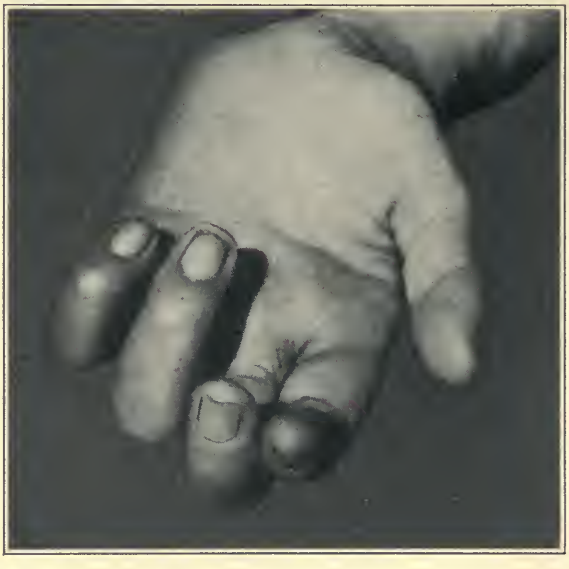
He can flex the forearm upon the arm so as to touch the hand to the face, chiefly by the action of the supinator longus muscle. He can extend the hand on the forearm better than he can perform any

FIG. 8



Photograph taken in December, 1905, showing the voluntary power in flexion of the fingers of the left hand.

FIG. 9



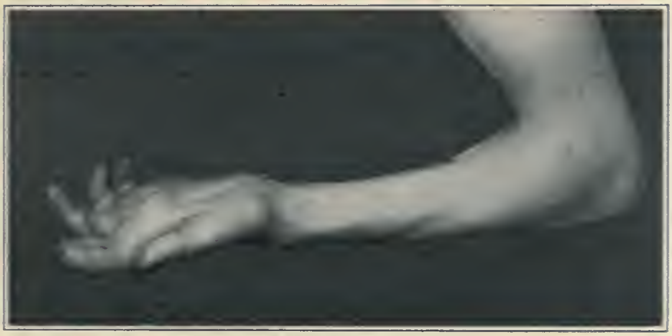
Photograph taken in December, 1905, showing extension of the fingers of the left hand. Extension is greater when more effort is made, as shown in Fig. 10.

other movement. (The musculospiral nerve was not cut transversely.)

Dr. C. S. Potts made an electrical examination November 7, 1905, and his report is as follows:

Right Arm. Deltoid, triceps, extensors of wrist, supinator longus respond well to the faradic current. The biceps responds, but not so well. In the median and ulnar distribution there is no response to the faradic current. AnClC greater than KClC (KClC very weak) marked modal change. The same maintains for the left arm, excepting that the deltoid does not respond as well to the faradic current as the muscle on the right side does. Owing to the corresponding movements of the arms it is practically impossible to pick out the individual muscles with any more accuracy than is stated above.

FIG. 10



Photograph taken in December, 1905, showing still more extension of the fingers of the left hand than is shown in Fig. 9. The index and middle fingers are doubled, as they were not held quietly while the photograph was taken.

REMARKS BY DR. FRAZIER.

The central end of an efferent fibre can make functional connection with the peripheral end of any other efferent fibre of the same class, whatever be the normal actions produced by the two nerve fibres.[1] Such is the physiological law confirmed by repeated investigations first undertaken by Flourens in 1824, upon which is based the principles of the operative treatment of palsies by nerve transplantation.

This principle is applicable to all forms of paralysis—to infantile spinal paralysis, to facial paralysis, to the paralysis due to traumatism of the peripheral nerves. Having proven beyond question of doubt the propriety of practising nerve anastomosis upon selected cases of infantile paralysis, there remains to be considered from the surgeon's point of view the mode of procedure. Several methods have been proposed and with each method the results have been variable. Too few cases have as yet been reported to enable one to deduce any conclusions based upon positive clinical data. The advantages which may be claimed for one method or another must be based chiefly upon theoretical grounds. These deal chiefly with the biological problems concerned with the regeneration of nerve fibres. Inasmuch as, at the present time, two so radically different theories as to nerve regeneration are entertained, it is not to be wondered at that different views should be held by surgeons as to the best method of effecting the anastomosis. These are:

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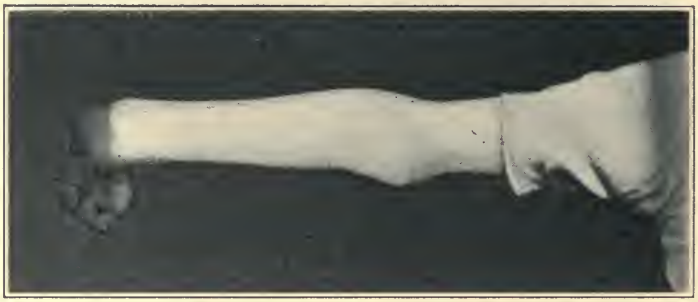
[1] Langley and Anderson. Journal of Physiology, 1904, vol. xxxi.

1. Lateral anastomosis of the peripheral segment of the nerve to the paralyzed group into the nerve supplying the unaffected group of muscles. (See Fig. 13, No. I.)

On theoretical grounds at least it may be assumed that the best results will follow an end-to-end anastomosis. No matter which theory one accepts, the central or peripheral, the best opportunity for one set of nerve fibres to unite with another is afforded by bringing into apposition the free ends of one with those of the other. When applied to the treatment of infantile palsy there is a strong objection to this method. We must bear in mind that there has been a certain percentage of failures, and one hesitates therefore to adopt a procedure which may aggravate rather than improve the condition existing prior to operation. Thus for example, if we split off from the unaffected nerve a flap with which to establish an end-to-end anastomosis with the affected nerve, we are robbing the healthy group of muscles of a portion of its nerve supply and may thereby permanently weaken that particular group. Dr. Spiller and I have therefore preferred the lateral anastomosis; that is, implanting the peripheral segment of the affected nerve into the unaffected nerve where the paralysis is not extensive, recognizing and acknowledging the possibility of failure in a certain percentage of cases.

Fig. 11



Photograph taken in December, 1905, showing the ability to raise the right upper limb. The operation on this limb was performed June 13, 1905.

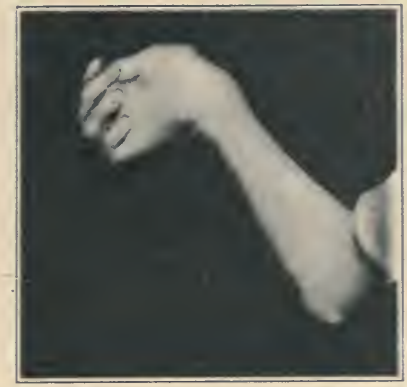
2. End-to-end anastomosis of the peripheral segment of the affected nerve with the central end of a segment split from the unaffected nerve. (See Fig. 13, No. IV.)

Should, however, with the perfection of the technique and with additional clinical experience, it be clearly demonstrated that there is sufficient assurance of restoration of function to the paralyzed muscles to warrant such interference with the nerve supply of the healthy group, this would be the operation of choice, and this or the operation following would probably be necessary where the paralysis is in several muscles.

3. Lateral anastomosis of the central end of a segment, split from the unaffected nerve into the affected nerve. (See Fig. 13, No. II.)

My attention was first called to this method of establishing an anastomosis by Hackenbruch.[1] Referring to Fig. 13, Nos. I. and II., it will be seen at once that one method is exactly the reverse of the other. Believing in the central rather than the peripheral theory of regeneration, Hackenbruch thinks this is the more logical mode of procedure. He believes that there would be a very much greater opportunity of functional union between the affected and the unaffected nerves if a flap from the unaffected nerve be engrafted into the affected nerve, since by this plan the young axis cylinders which sprout out from the central stump will almost surely find their way into the fibres of the paralyzed nerve. If, on the other hand, the paralyzed nerve is cut and grafted into the healthy nerve, accepting as he does the central theory of regeneration, he believes it more or less a matter of chance whether the new axis cylinders of the healthy nerve will find their way into the fibres of the paralyzed nerve.

FIG. 12



Photograph taken in December, 1905, showing the flexion of the right forearm on the arm.

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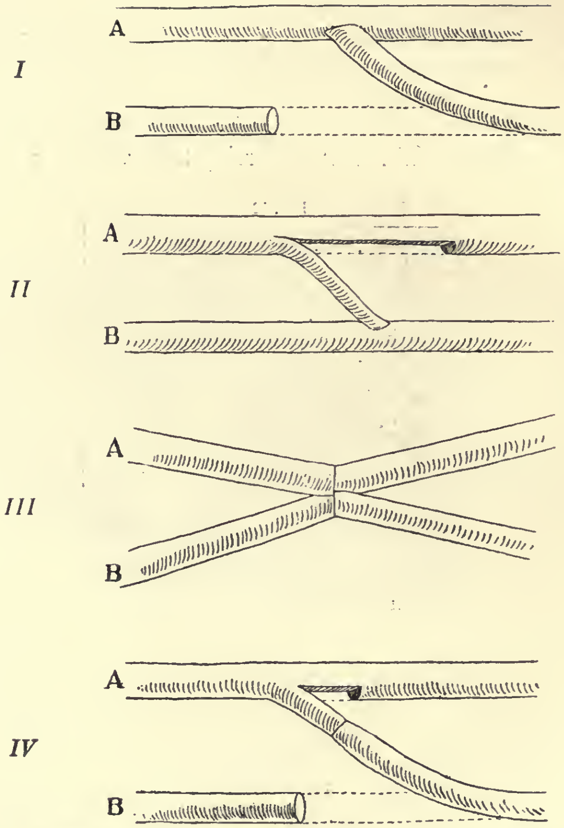
[1] Loc. cit.

4. End-to-end anastomosis of both central and peripheral ends of the affected nerve into the split segments of the unaffected nerve. (See Fig. 13, No. III.)

Dr. Spiller has already referred to Basil Kilvington's investigations. The latter, as a result of his experience with dogs, only recommends an end-to-end anastomosis using both peripheral and central segments of the affected nerves, on the ground that there may be a few fibres in the affected nerve which are functionally active and should not therefore be sacrificed. This operation might be applied with reason to those cases in which there is only partial and not complete paralysis in the affected group. The suggestion is not a practical one, however, since one would hesitate to recommend an operation where there was only partial disturbance of function. As to his recommendation that both the affected and

Fig. 13



Diagrammatic representations of various modes of anastomosis. *A* represents the unaffected, *B* the affected nerve.

unaffected nerves be divided before the anastomosis is affected, Spiller says this hardly seems a wise procedure. I would go still farther and call it distinctly an unwise procedure.

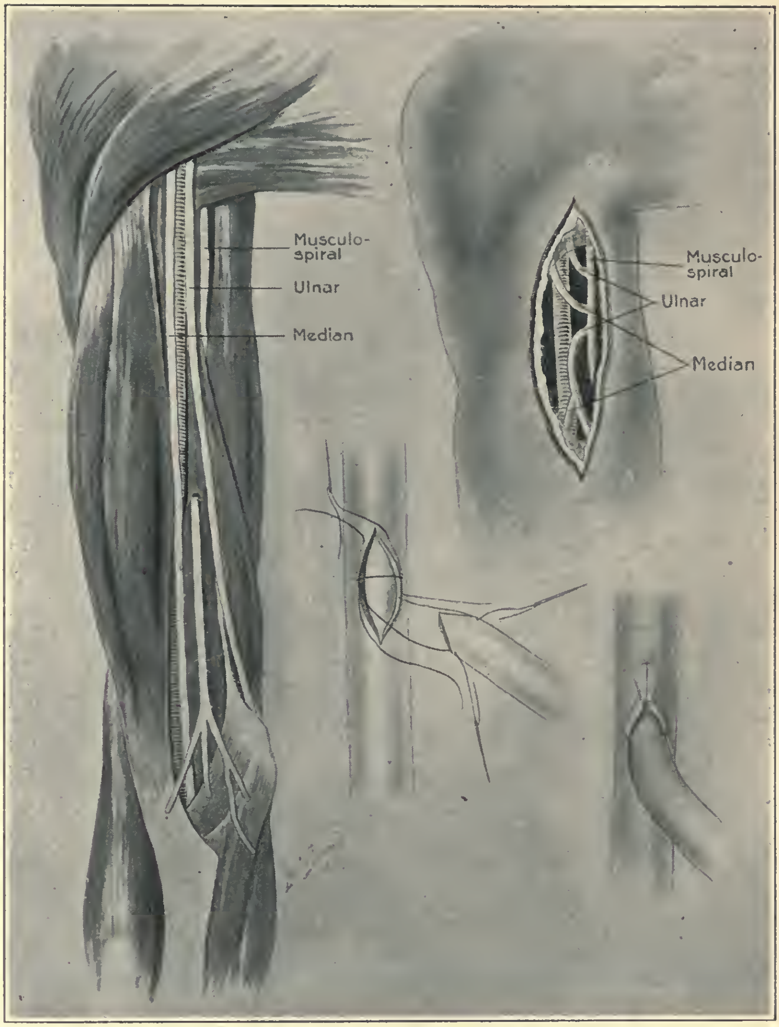
5. Lateral anastomosis of one or more muscular branches of the affected into the unaffected nerve.

This method is applicable of course to those cases in which but a single muscle is affected. Thus in a patient of Dr. Spiller's (case unpublished) the paralysis was limited to the tibialis anticus. The other muscles of the extensor group were not affected. Under such circumstances it is manifestly unwise to tamper with the nerve supply of the entire group. Accordingly the three minute branches supplying the tibialis anticus were divided and implanted into the musculocutaneous.

Once having determined upon the general plan of procedure, there remains to be considered the details of the technique. These will be taken up in the discussion of the operation for athetosis.

The principles underlying the application of nerve anastomosis in the treatment of athetosis have been referred to by Dr. Spiller. The first operation was performed March 16, 1905. An incision 4½ cm. long was made along the course of the brachial vessels and nerves in the upper half of the left arm. The ulnar nerve was exposed and freed from the areolar tissue throughout the length of the incision. The median nerve was exposed and isolated above the point at which it crosses the brachial artery and the musculospiral nerve for an equal distance. After the introduction of four sutures about 1 cm. apart into the ulnar nerve, two on either side, the nerve was divided between the two sets of sutures and the proximal and distal ends respectively implanted into the musculospiral. Four sutures were introduced in a similar manner into the median nerve, and after the nerve was divided the central end was implanted into the musculospiral nerve and the distal end was implanted into the ulnar nerve. The latter step was adopted because it was feared that too much traumatism might be inflicted upon the musculospiral if each of the four ends of the divided median and ulnar nerves were attached to it. The distal end of the median nerve was attached therefore into the distal segment of the ulnar nerve. The precise manner in which these anastomoses were made is graphically depicted in Fig. 14. At the points of anastomosis the nerves were enveloped with Cargile membrane, the wound closed with interrupted silkworm-gut sutures, and a plaster-of-Paris bandage applied over the dressing, binding the arm to the side. Despite the application of the fixation dressing it was not possible to keep the limb absolutely quiet owing chiefly to the frequent and violent contraction of the biceps muscle, whose nerve supply had not been interfered with. The seat of operation was subjected to so much disturbance through the uncontrollable activity of this muscle that I feared the process of union and regeneration might be seriously interfered with. Accordingly on April 17th, under my direction, Dr. Van Kaathoven performed upon the same arm a second operation designed to regulate the movements of the biceps muscle. An incision about 10 cm. long was made along the line of the axillary vessels and brachial plexus. The pectoralis major muscle was partially divided in order to

FIG. 14

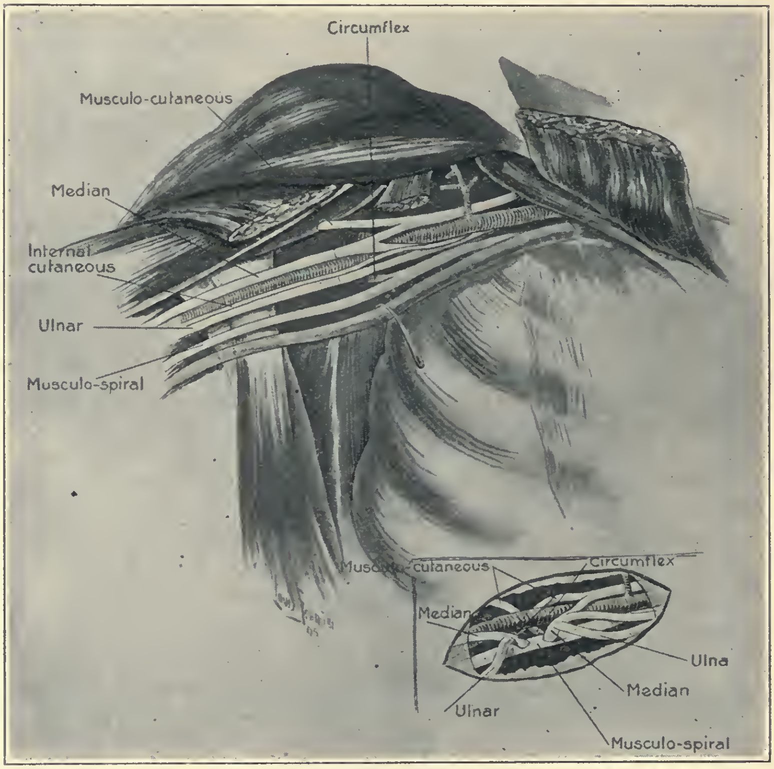


(*a*) To the left a sketch of the normal anatomical relations. (*b*) To the right and above, the manner in which the anastomoses were made in the operation on the left arm. (*c*) To the right and below, the introduction of sutures for a lateral anastomosis, two anchor sutures and a mattress suture.

afford a freer exposure. The circumflex and musculocutaneous nerves were isolated, their identity tested with an electric current, and two pairs of sutures introduced into each nerve; the nerves were divided between each pair of sutures and an end-to-end anastomosis effected in the following manner: the central end of the musculocutaneous was sutured to the peripheral end of the circumflex nerve, and the central end of the latter sutured to the peripheral end of the former. The nerves at the point of anastomosis were enveloped in Cargile membrane, the wound closed, and the arm bandaged firmly to the side. On June 13, 1905, encouraged by the results of the operation on the left arm, it was decided to carry out very much the same procedure in the right arm. The performance of this operation I entrusted to Dr. Van Kaathoven. An incision about 7 cm. in length was made over the brachial and axillary vessels, dividing the skin, superficial fascia, and about 2 cm. of the pectoralis major muscle. This incision exposed the vessels and brachial plexus. Each nerve was freed in turn. The musculocutaneous and circumflex were divided, and subsequently sutured in the following manner (see Fig. 15): the proximal portion of the circumflex into the distal portion of the musculocutaneous, and the distal portion of the circumflex into the proximal portion of the musculocutaneous. The median and ulnar nerves were then divided and a lateral anastomosis effected between the distal portion of the median and the musculospiral and between the ulnar and median. Similarly on the proximal side the proximal ends of the median and ulnar were united by lateral anastomosis. The muscle wound was closed with a double row of catgut sutures, the skin wound with interrupted silkworm sutures. A piece of rubber tissue, as a drain, was inserted into the apex of the axilla.

TECHNIQUE. Too much stress cannot be laid upon the importance of paying the strictest attention to details in the performance of operations dealing with the cranial or peripheral nerves. The nerves themselves must be subjected to a minimum degree of traumatism, otherwise the process of regeneration will be materially interfered with. To this end the nerves should never be grasped with forceps. In order to avoid the necessity of steadying the nerve while the suture is being introduced, we make it a practice to insert the suture before the nerve has been divided. (See Fig. 16.) If this precaution is taken it will not be necessary at any stage of the operation to grasp the nerve with forceps. When a nerve has to be divided it is very much better to use a sharp knife than a pair of scissors; the latter necessarily pinches and bruises the nerve fibres. It is scarcely necessary to call attention to the impropriety of including in the sutures any portion of the nerve but the sheath. If any nerve fibres are included in the suture these fibres will be so squeezed and crushed when the suture is tied that they will undergo degeneration. Anything which excites the formation of an excess of cicatricial tissue in or about the site of the anastomosis should be scrupulously avoided. The success of the operation depends: first, upon the nicety with which the apposition is effected and maintained; and secondly, upon the amount of cicatricial tissue at the site of anastomosis. The freer the nerves are at the point of anastomosis from the invasion of scar tissue the better will be the result. Everything which excites the tissues to more or less intense reaction, and therefore to the development of scar tissue, is a menace to the operation. Thus, wound infection is almost invariably fatal to success. The dissection should be as delicate as possible, so that a minimum degree of traumatism be inflicted upon the tissues; the incision should be made preferably to one side or another and not directly over the course of the nerve. The minimum number of sutures should be used, and after the anastomosis has been effected the nerves should be enveloped with Cargile membrane to prevent the invasion of scar tissue. As to the technique to be

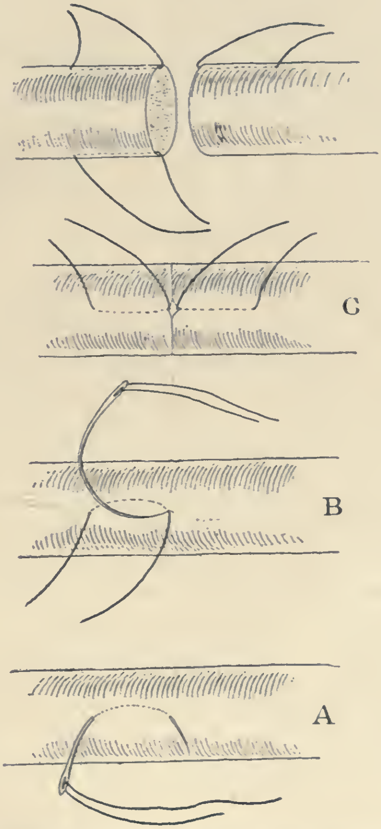
FIG. 15



(*a*) Showing normal relations of nerves and vessels. (*b*) To the right and below, a sketch showing the way in which the anastomosis was effected in the operation on the right arm.

observed in the introduction of the sutures, this will depend somewhat upon the way in which the anastomosis is to be made. In an end-to-end anastomosis two or three sutures will suffice to maintain apposition; in a lateral anastomosis the sutures should be introduced in the way illustrated in Fig. 16. Before dividing the nerve two sutures are introduced, one on either side only through the sheath. These sutures, when passed through the sheath on either side of the slit in the undivided nerve, are tied and serve to anchor one nerve to the other. In order to close the slit in the sheath and also to prevent the tip of the nerve from becoming disengaged a third suture is needed. To meet both these indications I have found a modified mattress suture very satisfactory. (See Fig. 15.)

FIG. 16



A, *B* and *C*, showing the way in which the sutures are introduced before the nerve is divided *D*, showing the nerve after division with sutures in place.

After the dressing is applied the limb should be encased in a plaster cast in order to ensure absolute rest, and I think it advisable to keep the limb perfectly quiet for four to six weeks. At the end of this period the cast is removed and the patient is then allowed to use the limb. The success of these operations depends in large measure on the faithfulness with which the after-treatment is carried out. Massage, galvanism, and prescribed exercises must be insisted upon and continued for six months to a year—continued as long as the condition is improving.

RESULTS. Whether or not the results of this operation will prove that an equalization of power or force between the two groups of muscles—the flexors and extensors—will be established remains to be seen. It will be interesting to note what influence the innervation of the musculospiral nerve will have upon the muscles supplied by the ulnar and median, and *vice versa,* what influence the innervation of the median and ulnar nerves will have upon the muscles supplied by the musculospiral. What will be the effect upon the extensor group of the transmission of the stronger impulses from the central segment of the median and ulnar nerves into the musculospiral nerve? Will these same impulses be diverted again from the musculospiral nerve into the peripheral segments of the median and ulnar nerves, or will there be no direct communication between the fibres of the proximal segments of the median and ulnar nerve with those of the distal segments? What would be the effect of stimulating electrically the central segment of the median or ulnar nerves? Would we get response in the distribution of the musculospiral nerve alone or in the median and ulnar nerves too? These are some of the interesting questions that suggest themselves, and no doubt a number of instructive phenomena concerning the transmission of nerve impulses and nerve regeneration may be observed in the case upon which we have operated. That one nerve can perform the function of another when both are physiologically united is a matter of common knowledge. Thus, for example, in cases of facial palsy we know that after anastomosis of the facial nerve to the hypoglossal or spinal accessory nerve either of these nerves may transmit impulses directly to the muscles supplied by the facial nerve, as well as to those muscles over which they originally presided. That the cortical centres of one nerve may be educated to preside over those of one or more is also an accepted phenomenon. Taking these physiological laws into consideration, it has occurred to me that in case the present plan of anastomosis which we have adopted in the treatment of athetoid movements is not successful, we might go even further than Dr. Spiller has suggested. Thus, for example, we might cut off entirely the connection of the nerve supply of the flexor group by permanently disconnecting the central segments of the median and ulnar and relying entirely upon the musculospiral nerve innervating the flexor group through its anastomotic connection with the distal segments of the median and ulnar nerves.

As to the results which have actually been obtained in the patient upon whom we have operated, we are able to make a favorable preliminary report. After recovering from the paralysis immediately following the operation the patient is regaining power in the distribution of the flexor group, and, as will be seen in Figs. 4, 5, 6, and 7, he is now able in the left extremity to voluntarily flex the fingers, the wrist on the forearm, and the forearm on the arm. At the last examination, November 6, 1905, the following notes were made by Dr. Spiller:

Left Upper Limb. There is still some stiffness of the entire limb, but this is not always present. The limb jerks very little when the man wishes to hold it quietly, and is not excited, but sometimes there is slight jerking on voluntary movement. When the left hand rests on some support he can almost fully extend the first finger and can extend the other fingers partially. He can partially flex all the fingers and can flex the wrist almost to a right angle, the power being greater on the ulnar side. He can flex the forearm on the arm to about the normal extent, and can partially raise the arm at the shoulder. Sensations for touch and pain are normal in all parts of the left upper limb, except in the palm of the hand, where there is still much impairment in the ulnar distribution. He cannot approach the thumb to the little finger. Muscular atrophy is slight.

Right Upper Limb. He still has athetoid movements at the shoulder, but they are apparently entirely or nearly entirely in the latissimus dorsi and muscles over the scapula, as the arm is drawn downward and backward during the spasmodic movements. He has slight return of power in the flexors of the fingers, but has much power in the extensors of the wrist. He can flex the forearm to a right angle with the arm and elevate the arm to a right angle with the trunk. He has much less involuntary jerkings at the right shoulder when he is not excited. Sensations for touch and pain are normal in the limb, except over the anterior aspect of the forearm and in the ulnar and median distributions of the hand.

There is therefore partial flexion and extension of the fingers of the left hand, much power in flexion and extension of the left hand on the forearm, and of the left forearm on the arm, and in elevation of the left arm at the shoulder. These movements are made with difficulty.

The return of power in the right upper limb is not yet so good as in the left, but this is because the operation on the right limb was performed more recently. The athetoid movements at the shoulder are in muscles that have not been operated upon. The voluntary movement present in the right limb is shown by slight flexion of the fingers, extension of the hand on the forearm, flexion and extension of the forearm on the arm, and elevation of the arm ta the shoulder.

The electric reactions are reported in the case history.

Reprinted from the University of Pennsylvania Medical Bulletin,
March, 1906.

A STUDY OF TWO CASES OF SYRINGOMYELIA, WITH NECROPSY.

One with Subdural Cyst, Hydrocephalus, and Optic Nerve Degeneration.

By Henry S. Hutchinson.

(From the Ayer Clinical Laboratory of the Pennsylvania Hospital, and from the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania).

The pathological findings in the two following cases of syringomyelia offer a few features which may be of interest. Both cases are called syringomyelia, as each seems to present those essential features which are supposed to distinguish syringomyelic from other kinds of cavities in the cord.

Syringomyelia, as it is now conceived, is not a distinct pathological entity, and cases have lately been reported in which the authors seemed in doubt what term to apply to the process described. The essential feature seems to be the presence in the gray matter of the cord, usually posterior to the central canal, of one or more cavities, showing evidences of progressive development, and associated with glia-hyperplasia and with changes in the neighboring blood-vessels. Most authors include the glia-hyperplasia in their conceptions of the pathological process; in Schlesinger's definition of syringomyelia, however,[1] the glia-hyperplasia is merely "often present." A cavity caused by distention, of the central canal is sometimes known as a hydromyelia, but from many cases in the literature it would appear that the process leading to this distention is often identical with that, so far as it is understood, leading to a syringomyelia, and the latter name is applied by many to both kinds of cavities.

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[1] Die Syringomyelie, Vienna, 1902.

The first case is reported through the kindness of Dr. Morris J. Lewis, who will later publish a full account of the clinical findings, etc. The pathological material was very kindly given me by Dr. Warfield T. Longcope, who performed the autopsy.

January, 1906. W. C., male, aged fifty years, white, married, childless. Contracted syphilis at age of twenty (1875), alopecia followed. Said to have been on "mixed treatment."

In 1890 he began to have headaches, and slight weakness in left leg, and shortly after this cerebral vomiting and transient tonic convulsions appeared.

These convulsions varied greatly in character, affecting either one side or other of the body, apparently indiscriminately, and were accompanied by unconsciousness. The first one seen (in 1890) was as follows: Arching backward of the head, dilatation of the pupils, turning of the head strongly to the left, with twitching of the *left* arm and leg, followed by transient paralysis of the muscles of the *right* side of the face, while another, occurring shortly afterward, affected the right arm and leg.

Choked disks were found, with hemorrhages into nerve head and retina with *complete, bilateral, temporal hemianopsia.* Sensation was blunted on the left side of the body. The knee-jerks were alike and normal. The urine contained no albumin. Under iodide of potash and mercury he rapidly improved, the convulsions vanished, and the choked disks, hemorrhages, and hemianopsia gradually disappeared. In one month's time the form fields had approached their normal shape, although concentrically contracted, the left the more so.

Although the knee-jerks now showed exaggeration, he felt so well that he was able to go upon a hunting trip that required much walking and horseback riding.

Early in 1891, however, convulsions again made their appearance; these were epileptiform in nature, and were accompanied by an olfactory aura, and the left leg became more feeble. By autumn this leg became paretic, and he had a hemiplegic walk. It was now found that there was marked anæsthesia from the lower border of the ribs downward, and by the spring of 1892 there was almost complete anæsthesia from the nipple line downward, and control of the bladder and rectum was lost. During this whole period he had been taking iodide of potash, in large doses, daily. Complete paraplegia ensued.

A period of quiescence, lasting at least ten years, now came on, during which time he was able to be about in a wheel-chair and take pleasure in his surroundings in the country. He was not seen again until October, 1904, when he returned to Philadelphia, and almost immediately contracted typhoid fever, for which he was treated in the Pennsylvania Hospital. In consequence of his paraplegia, frightful bed-sores made their appearance. He recovered from the typhoid fever in about five weeks, but the bedsores refused to heal, and he died a few weeks later, after returning to his home. During this period no change in the anæsthesia or paraplegia previously described could be detected.

The autopsy was performed by Dr. Longcope, June 24, 1905.

Anatomical Diagnosis. Compression of spinal cord in upper thoracic region, with subdural cyst; syringomyelia; moderate hydrocephalus; chronic leptomeningitis; chronic aortic endocarditis—two aortic leaflets; cardiac hypertrophy; chronic interstitial nephritis; acute bronchopneumonia; acute fibrinous pleurisy.

The body is emaciated, 178 cm. in length, with marked rigor mortis. Pupils equal. External genitalia apparently normal. Œdema of feet. Large, deep ulcer over sacrum and innominate bones.

Brain. The hardening fluid has affected the brain as the other tissues. Brain is of medium size. Dura not especially thickened; is not adherent to pia. Pia is whitish and more or less opaque, especially over the cortex, showing some general, rather streaky thickening. Convolutions well marked; sulci rather deep. Vessels at base show very slight thickening. The floor of the third ventricle bulges just below the optic chiasm. On section, the lateral ventricles are seen to be dilated, appearing several times the normal size. The right anterior horn is more distended than the left. On the left side the head of the caudate nucleus and the optic thalamus are well formed. On the right side the head of the caudate nucleus is much shrivelled, and the optic thalamus is also partially shrivelled. When a section is made through the upper part of the internal capsule on the right side an old cyst is found, occupying a large portion of the putamen of the lenticular nucleus, but extending also somewhat into the globus pallidus. The ependyma of the lateral ventricles looks thick and white. Several small cysts are seen in the choroid plexus of each lateral ventricle. The third ventricle is slightly dilated. The foramina of Monro (each) measure 7 mm. in diameter. The aqueduct of Sylvius is enlarged. Fourth ventricle is also large.

Spinal Cord. In upper thoracic region, before the dura is opened, there appears an enlargement. On cutting through the dura in the upper cervical region this membrane is seen to be quite thick, a little lower down it is found firmly adherent to the pia. The upper cervical region of the cord is fairly well preserved, but a little lower down the cord is seen to be somewhat flattened anteroposteriorly. Below the seventh cervical segment this flattening becomes very marked, and on the anterior aspect of the cord, where the dura is open, a definite cavity filled with fluid is found between the cord and the dura. The dura at this point is very thick. The eighth cervical and first thoracic segments, at least, seem to be intact, but small; immediately below this point the cord is seen to be compressed from the front, until it is converted into an indefinite, crescent-shaped mass of tissue a few millimetres across. The cyst-like formation in the membranes is open above, but ends below in a blind pouch, 11 cm. below the fourth cervical segment. Below the cyst the cord is surrounded by rather dense tissue, 1 cm. in thickness, which is limited by the dura. Section made here shows two open, longitudinal canals in the posterior funiculi, each measuring about 2 mm. in diameter. A section made 5 cm. below this point shows one large open canal, filling almost the entire cord. At this point no increase of tissue beneath the dura is seen. Traced downward, this large canal is seen to run for some distance in the cord, gradually becoming smaller, and disappearing before reaching the lumbar region. As it decreases in size it becomes surrounded by a narrow white line. At different levels in this cavity, delicate, cobweb-like threads are seen traversing it.

No gross lesions in the eyes or optic nerves.

Microscopically. The right pyramidal tract in the pons is a little degenerated. In the medulla oblongata the right pyramid is a little degenerated in its inner portion. The vessels in the pons and medulla oblongata are greatly dilated.

Sections through the middle of the cervical swelling (Fig. 1) show intense degeneration of the columns of Goll, slight rarefaction of the columns of Burdach, and considerable degeneration in each lateral column, chiefly shown in the areas of the columns of Gowers and of the direct cerebellar tracts, but extending also far enough inward to implicate a considerable portion of the crossed pyramidal tracts. The degeneration of these latter tracts is evidently retrograde in character, as the sections examined are near the destroyed area of the cord.. The sections here show a distinct central canal, with round lumen, lined by tall, epithelial cells. A little lower in the cord no canal is seen, but a clump of

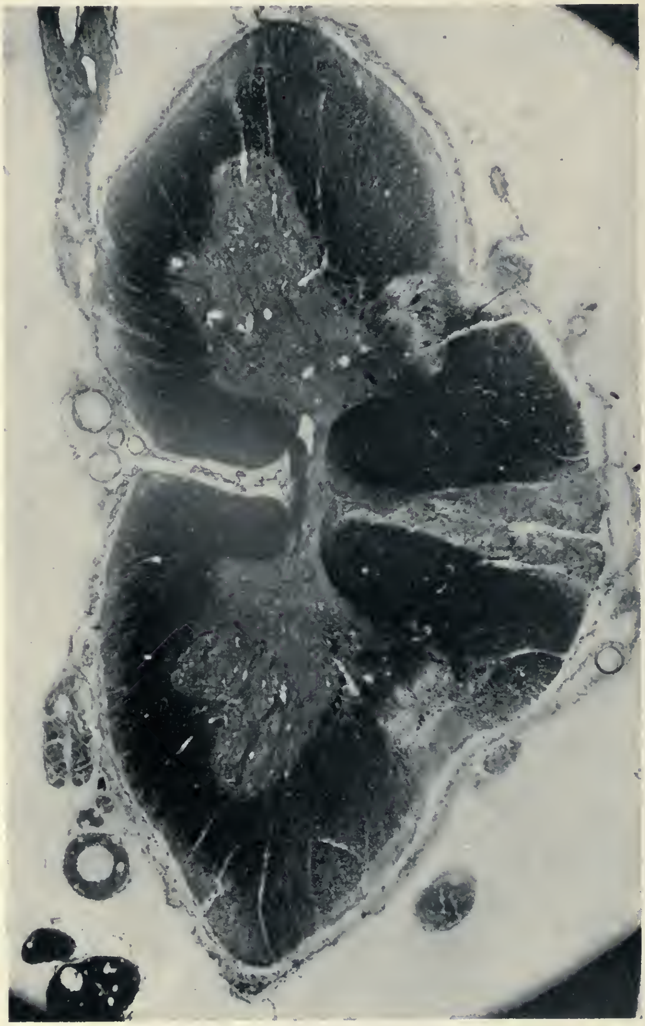

Fig. 1.—Case I. Cervical enlargement, showing retrograde atrophy of crossed pyramidal tracts.

epithelial cells appears. These transverse sections of the cord are smaller than normal.

First thoracic segment: Section is very small as compared with a normal cord. The degeneration of the lateral columns is much the same as that described above in the cervical region, but is more intense. The dura is very thick; the pia only slightly thickened.

Upper thoracic region, just above the point where the cul-de-sac between the dura and the cord ceases (Fig. 2), shows that the cord is greatly compressed, completely degenerated, and much flattened anteroposteriorly; that the pia and dura are greatly thickened, and that between the dura and the cord is a mass of loose, fibrous tissue. The dura is seen to be made up of compact lamellæ of fibrous tissue; the thickened pia appears as strands of fibrous tissue, more loosely arranged than those in the dura. There is a large, oval, cyst-like space in the membranes anteriorly. The tissue forming the wall of this space is

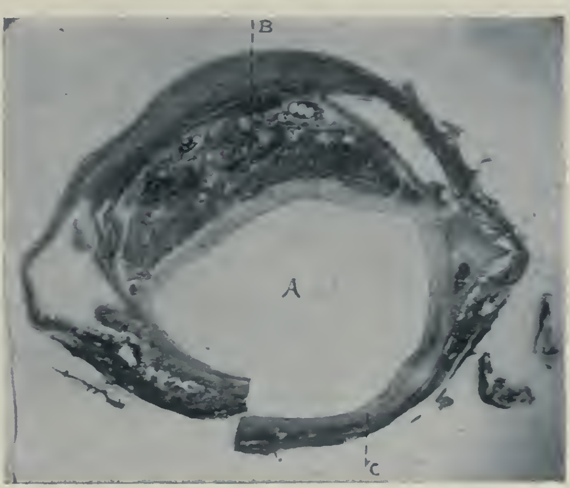

Fig. 2.—Upper thoracic region, showing subdural cyst (A), and, posterior to this, the necrotic cord (B). Anterior portion of greatly thickened dura at (C).

arranged in concentric, fibrous lamellæ. The nerve roots, cut in section, show much dilated bloodvessels, many of them with walls perfectly hyaline. No trace of central canal or ependymal cells seen.

Section at extreme lower end of the cul-de-sac (Fig. 3) shows that the cord at this level is not so compressed as in the section just described, but is as much degenerated. A small cavity, triangular in shape, is seen at the end of one posterior horn in the posterior columns, very near to the periphery. Between the dura and the cord, anteriorly, is seen a dense mass of fibrous tissue. A thick clump of cells occupies the site of the central canal.

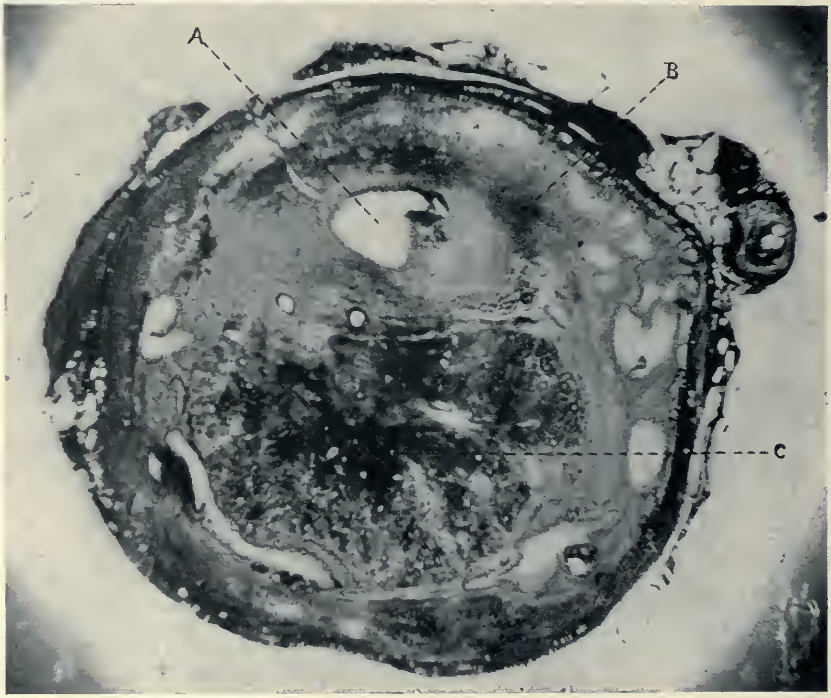

Fig. 3.—A few centimeters below section represented in Fig. 2. Extreme lower end of subdural cyst (A). Necrotic cord is shown at (C) surrounded by fibrous tissue (B).

A section a little lower in the thoracic region shows two distinct cavities, one occupying almost the whole of the columns of Goll and Burdach and the posterior horn of one side; the other occupying the posterior horn, and extending into the lateral and posterior columns on each side of this horn. These cavities do not communicate with each other, and, traced downward in the cord, each ends blindly. Each is replaced, however, by a new cavity in the corresponding posterior horn. One of these new cavities, traced downward, soon disappears; the other enlarges and forms the cavity described at the autopsy. Fig. 4 represents a section through the thoracic region a little below the level of that shown in Fig. 3. The normally stained nerve fibres are few, and chiefly gathered about the anterior horns and in the posterior portion of the posterior columns. The fibrous tissue between dura and cord is not so dense as at a higher level. Serial sections were made through the lower

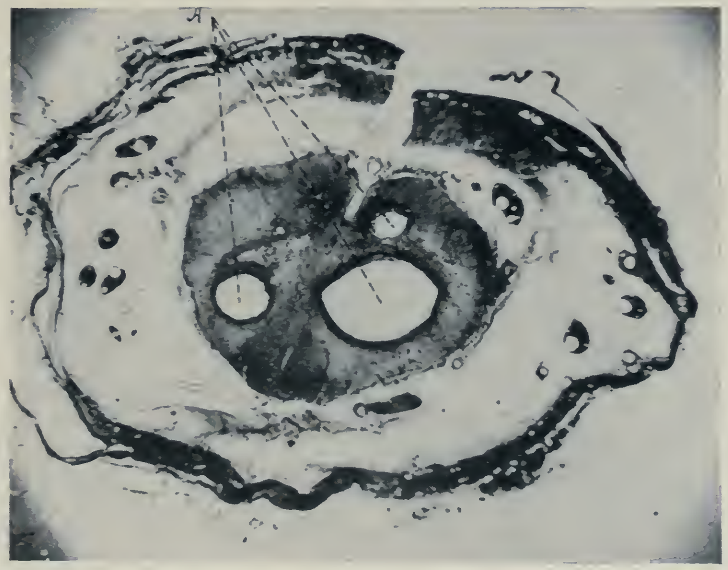

FIG. 4.—Slightly lower in the thoracic region. Cavities in the chord (A).

end of one of the smaller cavities, and continued into the beginning of the next cavity in the same horn; traced downward, the cavity gradually grows smaller, and, as it does so, is seen to be surrounded by a definite wall of thickened neuroglia. Before it has disappeared, a rarefaction of tissue in the gray matter just anterior to it can be seen. Traced downward this rarefaction becomes more marked, and finally gives place to a cavity. In the neighborhood of this cavity is a mass of thickened neuroglia. Each anterior horn, for a short distance, is also the seat of a small cavity, not surrounded by thickened neuroglia, and apparently the result of a rarefaction of tissue.

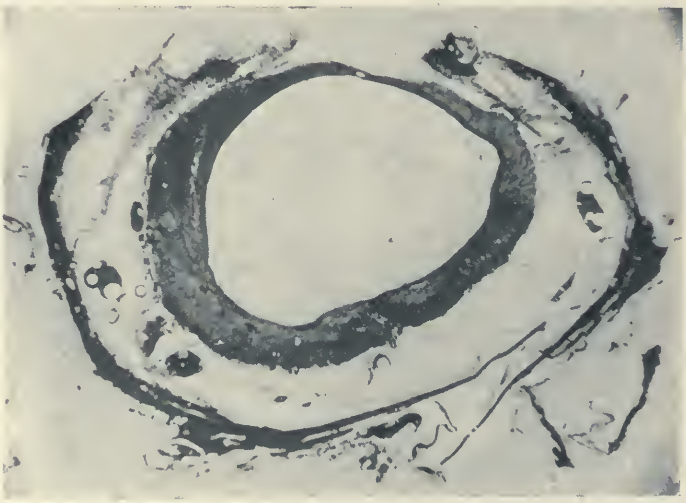

Fig. 5.—Middle thoracic region, showing a large cavity occupying most of the cord.

A little below this point only one cavity can be found (Fig. 5), and this occupies the whole of the anterior portion of the posterior columns, extending from one posterior column to the other, and greatly distorting the cord. The crossed pyramidal tract is degenerated on each side. This cavity, traced downward, grows smaller, and ceases in the lower thoracic region. Sections made immediately below the point where the cavity ceases, show, in the region corresponding to the cavity above, an area of glia-hyperplasia in which but few stained nerve-fibres are found. The thickening of the membranes, especially of the pia, is much less intense, and, where the cavity is very small, the pia shows only little clumps of thickened tissue, posteriorly and laterally.

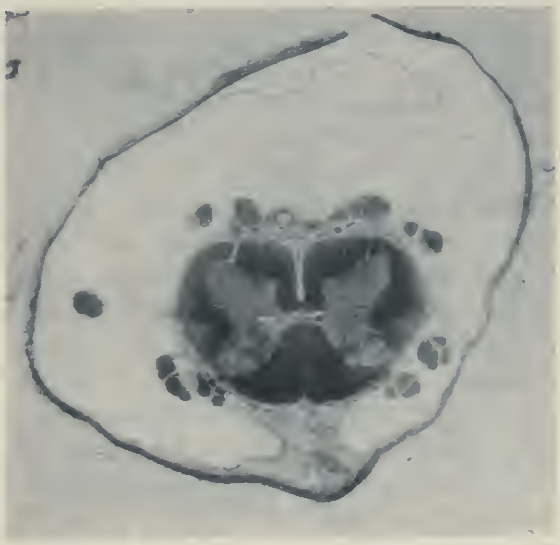

Fig. 6.—Lumbar enlargement, showing distention of subdural space, and retrograde atrophy (poorly shown) in the posterior columns. The crossed pyramidal tracts are degenerated.

Sections were made through one of the thread-like bits of tissue, which were described as passing from one side of the cavity to the other, traversing its lumen. Stains by fuchsin show a composite mass of fibres; stains by the Weigert hæmatoxylin method show a few black-stained nerve fibres, traversing the lumen of the cavity through the thread of tissue.

Below the point where the cavity ends, in the lower portion of the thoracic region and in the upper lumbar region, there is some sclerosis of the posterior columns (Fig. 6). This is evidently the result of a retrograde atrophy. The lumbar cord, except for secondary degeneration, seems to be normal. The subdural space is much larger than normal, and there was evidently an abnormal amount of cerebrospinal fluid about the lower portion of the cord.

A word about the walls of the cavities and the bloodvessels in the cord. Each cavity, where of good size, is seen to be surrounded by a layer of thickened neuroglia, arranged in strands of wavy lamellæ about the cavity. Toward the termination of one of the cavities, which was traced in serial sections, this lamellated appearance ceases; the wall of the cavity is here very irregular; necrotic tissue and even bloodvessels, transversely cut, project into the lumen.

The bloodvessels throughout the cord appear greatly dilated, and show in the sections as large, round holes. All present thickening of their coats, and the walls of many are perfectly hyaline, staining evenly red (fuchsin). A few appear to possess no lumen, showing as circular, hyaline masses, staining red. From the outer coats of many, strands of connective tissue are seen streaming off into the surrounding nerve substance; where the vessels are cut in longitudinal section this is plainly seen. Sections stained by Van Gieson's and also by Mallory's connective-tissue method show these strands fading into the nervous tissue; small portions of the walls of the syringomyelic cavities are seen to be lined by wavy strands of connective tissue. The regions of the syringomyelic cavities show some increase in the number of bloodvessels.

Optic Tracts and Nerves. Microscopic Sections. The left optic tract appears to be normal. The right optic tract is not so wide as the left, and as it approaches the commissure is much degenerated on its inner aspect. The left optic nerve is very degenerated, and especially on its inner aspect near the chiasm (Fig. 7). The pia about the chiasm is somewhat thickened, and presents a very slight, round-

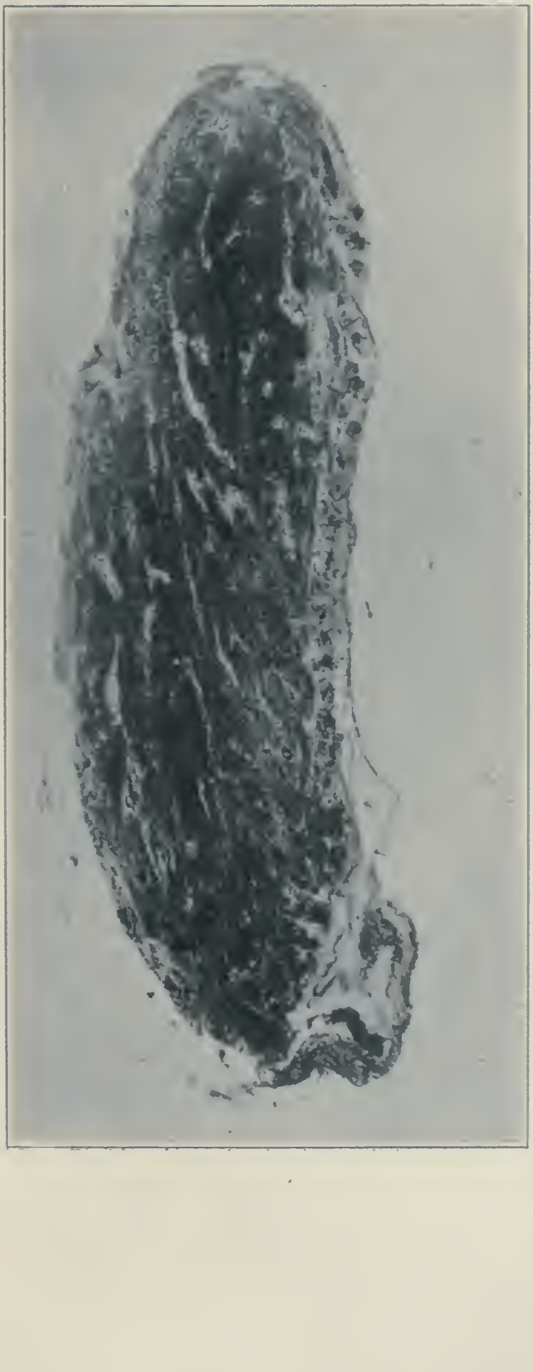

FIG. 7.—Left optic nerve. Degenerated fibres in inner portion of nerve.

cell infiltration. The right optic nerve (Fig. 8) is partially degenerated, especially in its central portion, but not so much as the left, and the fibres on the inner portion of the nerve do not appear to be more degenerated than those on the outer portion of the nerve.

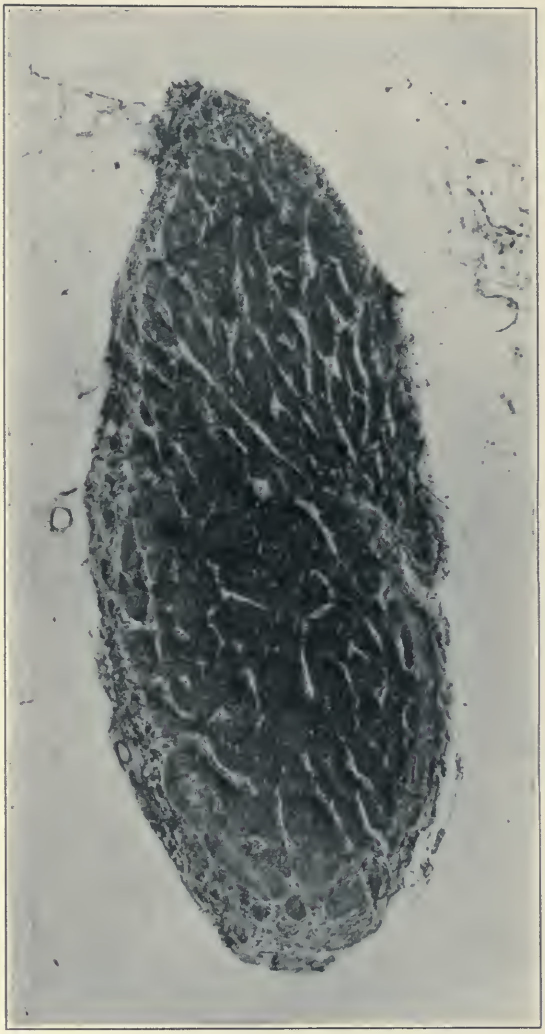

Fig. 8.—Right optic nerve. Some degeneration in centre of nerve.

The choroid plexuses of the lateral ventricles show, in the epithelium, a somewhat larger number of intracellular globules than normal. The bloodvessels are very dilated.

Microscopic examination of the other organs of the body confirms the anatomical diagnosis made at the autopsy. In addition, the liver shows advanced fatty degeneration.

For the pathological material of the next case I have to thank Dr. Charles S. Potts.

The case is as follows:

M. W., female, white, aged sixty-seven years, occupation cook. Entered Philadelphia Hospital in service of Dr. Burr, May 31, 1902. Hospital number, 698. Chief complaint "stroke." Family history: Father died suddenly, probably paralytic stroke; mother died of old age. Past medical history: had diseases of childhood; fourteen years ago had "stroke," and was in hospital two years and eight months. Was then able to walk only by using a chair. Present illness: had another "stroke" on the 24th of May, 1902, (one week ago); has been partially paralyzed on right side. Condition: woman past middle age, thin, and unable to walk. Pupils equal, and respond to light. Tongue coated quite thickly and yellowish white. Pulse regular, but weak. Arteries comparatively normal. Considerable depression above and below the right clavicle; percussion note duller over right than over left apex. Heart negative. Reflexes present. Right knee-jerk apparently absent. Left leg somewhat deformed from an old fracture.

A year later, on June 13, 1903, 4.15 P.M. Patient dull, and so far gone that it is impossible to obtain any definite answers. Grip of right hand is poorer than grip of left. There is wasting of thenar and hypothenar eminences, and of the interossei muscles, equally marked in the two hands. The biceps, triceps, and wrist reflexes of the right upper extremity are prompter than those of the left. Has apparently no wasting of the lower extremities. Patellar jerk and Achilles jerk absent in both lower extremities, some movement in the quadriceps muscle of the right leg being seen, however. Plantar irritation produces extension of large toes on both sides. Tongue is protruded to about the lips. Left pupil is excentric. Both pupils respond slowly to light. The movements of both eyes are normal. Patient has apparently no anæsthesia. For the last three weeks has had incontinence of urine and feces. No paralysis of the muscles of the face.

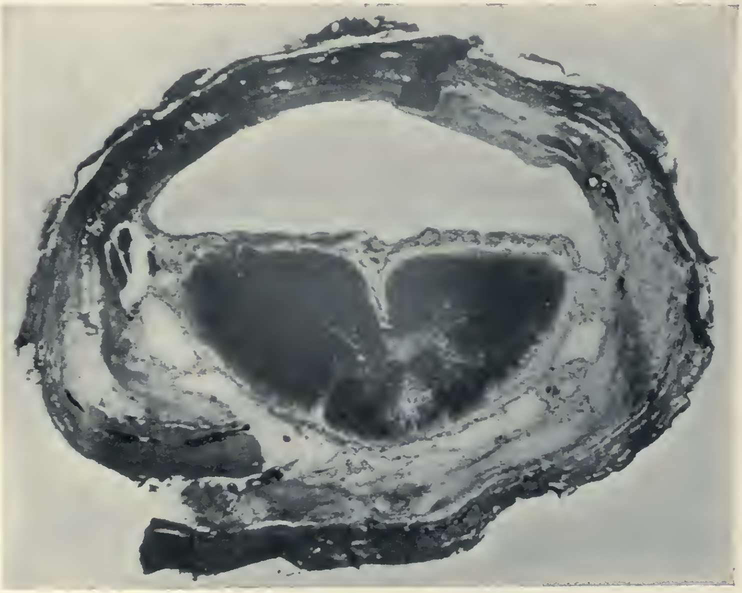

Fig. 9.—Case II. Upper thoracic region. Greatly thickened dura and adhesions between dura and cord. Rarefaction of posterior columns.

Primary Diagnosis: Right hemiplegia.

Complications: Œdema of lungs.

Patient died June 13; time of day not given.

Microscopic Examination. Sections were cut at different levels in the cord and medulla oblongata.

The medulla oblongata is normal by the Weigert hæmatoxylin stain. The walls of the bloodvessels are somewhat thickened. There is moderate round-cell infiltration of the pia, and about some of the vessels in the medulla oblongata.

In the cervical region the posterior columns are much degenerated, as are also the direct cerebellar tracts. The dura and pia are thickened, and in the posterior part of the cord are firmly united. On the anterior portion the dura is still separated from the pia. There is degeneration in the peripheral zone of the anterior columns as well as in the lateral columns. A round-cell infiltration is seen in the pia, and the vessels of the pia are much thickened. In the place of a central canal there is seen a clump of cells, without lumen, passing over irregularly into the surrounding tissue. Lower down, in the thoracic region (Fig. 9), the degeneration in the posterior columns is intense, and the whole border zone of the anterolateral columns is much degenerated. In the posterior columns the nerve tissue appears very rarefied, but in this tissue there are an extraordinarily large number of transversely cut bloodvessels, with thick walls. Adhesions between the dura and the pia are present at all parts, except in the anterior portions of the

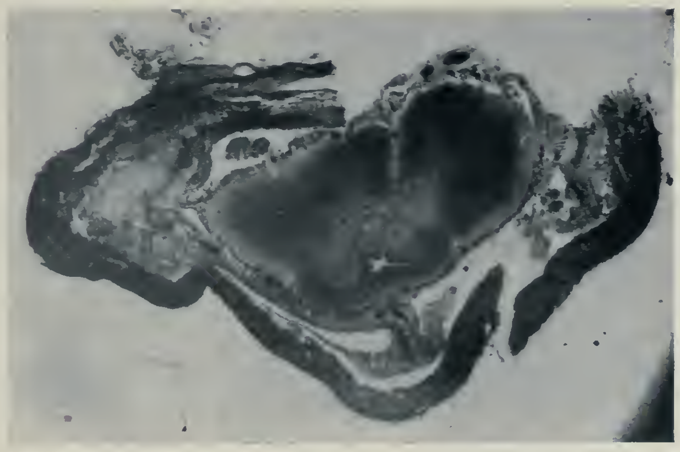

Fig. 10.—Lower than Fig. 9. Cavity seen in posterior columns.

cord, and the dura is greatly thickened. At the site of the central canal there is the same clump of cells without lumen; but posterior to this, and separated from it by neuroglia tissue, is another similar clump of cells.

A little lower (Fig. 10) in the thoracic region the thickening of the dura and the adhesions between the dura and the pia are slightly less intense, but a cavity is found, star-shaped, in the degenerated posterior columns, and in each posterior horn of the gray matter there is some rarefaction of tissue. At the site of the central canal a group of cells is seen, the most posteriorly situated of which are arranged about an irregular lumen. Section taken a little below this point shows the cavity in the posterior columns now in continuity on either side, with a cleft extending into the posterior horns of the gray matter.

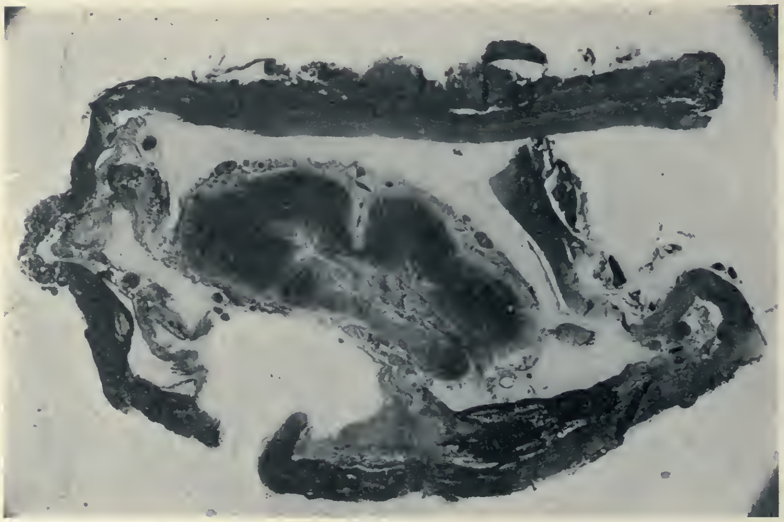

Fig. 11.—Slightly lower than Fig. 10. Notice great thickness of dura and adhesions to cord.

Still lower in the thoracic region (Fig. 11) the cavity is found to be larger, and to extend from the base of one anterior horn, across the posterior columns, to the posterior horn of the other side. The posterior columns and the border zone of the anterolateral columns are intensely degenerated. Some round-cell infiltration is found about the bloodvessels of the cord, and in the rarefied tissue surrounding the cavity. As in the sections above this point, the cavity presents distinctly the appearance of formation by rarefaction and absorption of tissue. The walls are very irregular, and bloodvessels project into the lumen, some of them appearing completely isolated in the cavity. About the cavity are many areas of homogeneous material, staining light pink (fuchsin). The cavity is nowhere lined by epithelial cells. The thickening and adhesions of the membranes are less intense than higher up in the cord. The group of cells at the site of the central canal is divided into two parts, an anterior and a posterior, the latter having an irregular lumen.

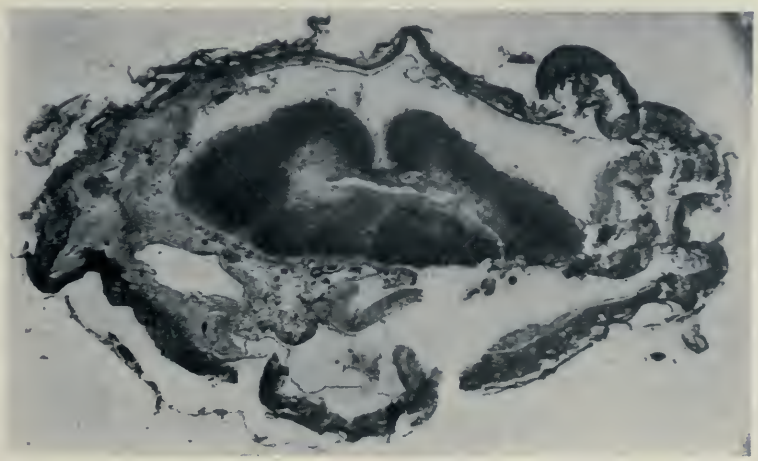

Fig. 12.—Thoracic region, lower than Fig. 11. Cavity in the gray matter.

At a little lower level in the thoracic region (Fig. 12) a cavity is seen, a short distance behind the site of the central canal, lined on its anterior and posterior aspects by epithelium. At its two lateral extremities the neighboring nervous tissue is markedly thinned out, and contains many thick-walled bloodvessels. As in Case I, strands of connective tissue are seen streaming off from the adventitia of the bloodvessels into the surrounding nerve tissue (Van Gieson's connective-tissue stain). The rarefaction of tissue in the posterior columns is no longer present, but this condition is now seen in the base of each posterior horn of the gray matter. The posterior roots are intensely degenerated; the anterior roots are more nearly normal. The thickening of the pia and dura is much less intense than higher up in the cord

Lower in the cord, but still in the thoracic region, the adhesion between the dura and the pia ceases, but there is still much degeneration of the posterior columns, the crossed pyramidal tracts, and the border zone of the anterolateral columns. No cavity in the cord is seen.

At about the twelfth thoracic level the degeneration of the cord is much the same as that described above, though less intense in all parts of the cord. The pia at this level is thickened, and very little infiltrated with round cells; the bloodvessels in the pia are thickened.

In the lumbar region the posterior columns are still partly degenerated, as are also the posterior roots. The anterolateral columns are no longer degenerated in the border zone, except near the posterior horns, where the area is occupied by the crossed pyramidal tracts. The ependymal cells of the central canal here appear in two groups, a right and a left, separated by neuroglia tissue. Neither group possesses a lumen.

RÉSUMÉ. Case I. Man, at age of twenty years (in 1875), contracted syphilis. Fifteen years later, headaches, vomiting, and convulsions appeared. The convulsions affected both sides of the body and were accompanied by unconsciousness. There was complete bilateral, temporal hemianopsia, and the ophthalmoscope revealed choked disks and retinal hemorrhages. Under anti-syphilitic treatment patient improved greatly, the convulsions left him, and sight returned. Less than a year later, however, the convulsions returned, and from the lower border of the ribs downward, first anæsthesia then paraplegia developed. For the next fourteen years, until the patient's death, there were no changes in the anæsthesia or paraplegia.

The anatomical findings were: In the upper thoracic region a large cyst in the membranes, anterior to the spinal cord, compressing and causing complete degeneration of the latter, and ending below in a cul-de-sac. Great thickening and adhesion of the membranes at and below this point. Lower in the cord, but confined to the thoracic region, several syringomyelic cavities of varying extent, one of them, the lowest, occupying the entire centre of the cord, having the cord about it as a narrow ring (Fig. 5). A moderate, internal hydrocephalus was present, and degenerations were found in the optic nerves, especially the left, and in the inner portion of the right optic tract. An old cyst was found in the corpus striatum of one side, and consequent degeneration of some of the pyramidal fibres below. The spinal cord, traced downward, showed more and more evidences of compression, until in the region of the cyst it appeared as a crescent-shaped, necrotic mass of tissue, surrounded by dense, fibrous tissue. Below this point it expanded again, to be occupied, however, by a series of cavities, the last and largest of these ending before the lumbar region was reached. These cavities, excepting a small one in each anterior horn, occupied the posterior horns of the gray matter. The lumbar region, except for retrograde degenerations in the posterior columns of the cord, appeared normal.

A distinct central canal was present in the upper cervical region, though it was surrounded by a dense mass of cells; below this point, throughout the cord, only a mass of cells without lumen, occupying the site of the central canal, were seen.

Case II. Woman came to hospital complaining of "stroke;" partially paralyzed on right side. Same kind of attack fourteen years before. Knee-jerk on right side absent; other reflexes, including pupillary, present. On day of death, a year later, knee-jerks on both sides absent, wasting of thenar and hypothenar eminences, and very weak grip in right hand. Eye movements normal, and no paralysis of facial muscles. Incontinence of urine and feces.

The anatomical findings were: A cavity of small extent, situated in the posterior half of the spinal cord, in the thoracic region. Above, this cavity appeared to originate in a rarefaction and absorption of the nerve tissue of the posterior columns; lower, where the cavity was somewhat more anteriorly situated, its anterior and posterior walls were seen to be lined by epithelium, while its two lateral extremities ended in thinned-out nervous tissue, rich in thick-walled bloodvessels. The membranes of the cord were greatly thickened, this being most marked at a level slightly above the cavity in the cord, where the membranes, posteriorly and laterally, were firmly adherent to one another. The pia showed some round-cell infiltration. The bloodvessels in the cord and membranes throughout showed thickened walls, and about some of them a slight round-cell infiltration was seen.

A possible connection between the several lesions in the first case suggests itself. The man had syphilis. This would account for the thickening and other pathological changes in the meninges. Where this process was most marked—in the upper thoracic region of the cord—all the meninges were found glued together. As a result of this, the spaces in the meninges above this point were no longer in communication with those below, and the flow of the cerebrospinal fluid was prevented. This fact would account for the cyst-like formation in the meninges, and for the great compression of the cord above this point. The prevention of the free circulation of the cerebrospinal fluid would cause a collection, under pressure, of the same in the cranium—over the surface of the brain and in the lateral ventricles. The consequent bulging of the floor of the third ventricle, with pressure on the optic tracts and chiasm, would account for the degenerations found in the optic tracts and optic nerves. That there was any direct connection between the pressure caused by the meningeal cyst in the thoracic region and the syringomyelic cavities found in the cord below this point is not likely. That one of these cavities, the largest, antedated the beginning of the cyst formation is very probable; the others may have originated later. As for the degener-

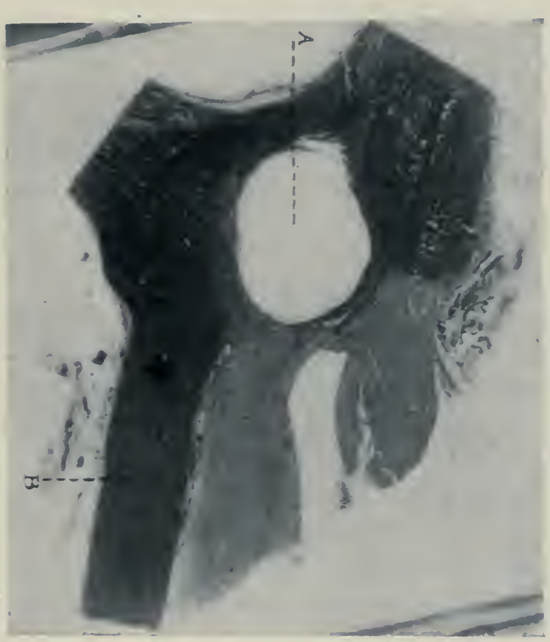

Fig. 13.—Normal optic chiasm, showing relation to third ventricle (A). Left optic tract at (B). It may be seen from this section how distention of the third ventricle exerts pressure upon the optic chiasm and optic tracts.

ations in the tracts, they were such as were to be expected from a complete destruction of the cord in the upper thoracic region.

The hydrocephalus, though its existence seems to be explained by the adhesions in the meninges of the cord below, may yet have some other explanation—in part, at least. Hinsdale[1] says that Kupferberg, in reviewing the literature, found out of 130 cases of syringomyelia, chronic idiopathic hydrocephalus present in fifteen. Couvelaire,[2] in 1899, reported a case of a man of thirty-six years, in whom there was great hydrocephalus and a cavity throughout the whole length of the cord; the cord was reduced to a mere shell. As for the degenerations in the optic nerves, these, as was remarked, are probably to be explained as a result of the hydrocephalus. Fig. 13 is a section through a normal optic chiasm, and shows the relations of the floor of the third ventricle to the chiasm. It will be seen from this that pressure may readily be exerted from the third ventricle upon the optic tracts and chiasm. The section was kindly lent to me by Dr. Spiller.

Weisenburg and Thorington[3] have recently reported a case of syringomyelia with double optic neuritis, in which they believe the cause of the latter to have been internal hydrocephalus. They have been able to find, in the literature, only two cases of optic neuritis as a complication of syringomyelia, one by Bullard and Thomas, and the other by Saxer. Both of these cases showed at autopsy marked internal hydrocephalus. Weisenburg (*The Eye and the Nervous System: their Diagnostic Relations*, edited by Posey and Spiller) speaks of the possible connections between syringomyelia and optic neuritis, and emphasizes the importance of a complicating internal hydrocephalus. Referring to primary optic atrophy, he says that this occurs less often in syringomyelia (unless complicated with tabes dorsalis) than in any other disease. He also speaks of the as yet unexplained relation between optic-nerve atrophy and disease of the spinal cord, and, reviewing the theories put forth to account for this relation, he concludes with Gowers and others that the optic-nerve atrophy is uniformly progressive with the spinal-cord symptoms, and that both are due to the same primary cause—a lack of vital endurance, or an "abitrophy" (Gowers)—picking out especially the optic nerve and the spinal cord for degeneration. The involvement of the optic nerve in myelitis he explains in much the same way—by the action of a toxin which picks out, besides the spinal cord, especially the optic nerve. The optic nerves, in the first case, were not uniformly affected, the inner half of the left optic nerve and the inner portion of the right optic tract being completely degenerated. The cause of this could not have been general, but must have been local, and is found, probably, in the distention of the floor of the third ventricle. The other degenerations seen in the two optic nerves, especially those in the central part of the right nerve, may have their explanation in some such general cause as those mentioned above. That the pressure of the third ventricle upon the optic chiasm did not produce a permanent loss of function of the crossed fibres of the two optic nerves is shown by the fact that the left optic tract was found intact.

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[1] Syringomyelia. Monograph. Philadelphia, 1897.

[2] Revue Neurologique, 1899.

[3] American Journal of the Medical Sciences, December, 1905.

The retrograde atrophy of nerve fibres seen in the cord in Case I. deserves to be mentioned. In the cervical region the crossed pyramidal tracts, and in the lumbar region the posterior tracts, showed here and there degenerated, or rather atrophied, fibres. The nature of this process, in which the central stump of an injured or cut nerve fibre becomes atrophic, is not at all clear. Several explanations have been offered, but no one of them is satisfactory. The literature on this subject is not large. Spiller[1] in 1898 reported a case in which he found retrograde atrophy in the pyramidal fibres above, and in the fibres of the posterior columns below the seat of the lesion. He was able to trace this process for a distance of 4 cm. from the lesion. Recently J. R. Hunt[1] has published an article on "Retrograde Atrophy of the Pyramidal Tracts," in which he interprets the phenomenon as due to the functional inactivity of the motor cells. He says that the sensory tracts are comparatively free from this form of atrophy.

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[1] Johns Hopkins Bulletin, 1898.

Mention was made of the fact that the epithelial cells of the choroid plexuses showed a somewhat larger number of intracellular globules than are normally found. Raubitschek[2] has recently studied the appearance of these globules and their occurrence in cases of meningitis. They give, as is known, the same reaction as fat with osmic acid, but can be differentiated from fat by special straining with pyronin. Petit and Girard[3] have found that they could experimentally produce a great increase in the number of these globules by administering muscarine and other drugs, causing, as they put it, a hypersecretion on the part of the cells of the choroid plexus. The occurrence in the present case of the globules in large numbers may be explained, perhaps, as the effect of some toxic substance in the blood or cerebrospinal fluid—possibly the same which caused the pathological changes in the meninges of the spinal cord, and, if their existence in numbers indicates a hypersecretion, they may stand in some relation to the hydrocephalus.

The mode of origin of the cavities found in the cords of these two cases is by no means clear. The etiology of syringomyelia is still somewhat obscure, and from the cases reported in the last few years, and from the work done on the subject, the causes of the lesion would seem to be very varied. In fact, nearly every writer on the subject has put forth some new view as to the possible origin of the disease in his case. From an examination of the different cavities found in the present two cases there would seem to be, in each case, certainly more than one process at work in the production of the cavities.

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[1] Journ Nerv. and Ment. Disease, N. Y., 1904.

[2] Zeitschr. f. Heilkunde, April, 1905.

[3] Comptes Rendus, Soc. de Biologie, 1901, 1902.

That most syringomyelias are congenital, or the result of some congenital malformation, is the view held by the majority of authors. Very many cases have undoubtedly this mode of origin: a faulty closure of the central canal in embryonic life, leaving, in the gray matter of the cord, posterior to the central canal, nests of epithelial cells which may later show a tendency to proliferate (Hoffman). But it is generally accepted that a typical syringomyelia may come into being in other ways than this. Vascular changes, especially hemorrhages; tumors—gliomata and sarcomata—reacting upon the surrounding tissue, and themselves degenerating; changes in the meninges, perhaps in relation with vascular changes, and other processes have all been held to account for the presence of syringomyelia.

The congenital abnormalities may be small, and perhaps lie dormant in the cord until some exciting cause comes to start a pathological process. The cavities would then come into existence by the proliferation of the nests of epithelial cells about a previously existing lumen, with destruction of surrounding tissue, or by a liquefaction and absorption following an irregular proliferation of these cells. The epithelium of the central canal itself is believed to show sometimes an inherent tendency to proliferate, forming buds and shoots ("ependymal adenoma"—Babes). The exciting cause of the proliferation of these nests of cells or of the cells of the central canal would be direct or indirect—direct, if through toxins or otherwise the cells themselves were stimulated—and indirect, if, through destruction and rarefaction of surrounding tissue, the proliferation of the cells took place because of decreased tissue resistance.

As to the nature of this exciting cause, there are several opinions held. Schlesinger says that the cells being situated in the centre of the cord are in the position where vascular changes will be the most felt, and that in cases where the pathological changes in the bloodvessels are not quite sufficient to cause a necrosis of the tissue they may yet be sufficient to awaken in the abnormally situated cells a tendency to proliferate. Rarefaction of peri-ependymal tissue due to poor nutrition may, according to Redlich, give rise to a "hydromyelia." Saxer believes that the multiplication of the cells in question is a non-inflammatory, autochthonous cell proliferation, and that the later formation of cavities is due to vascular changes. Vetlesen[1] regards the proliferation as a species of genuine tumor-formation, with extremely slow development; according to his view, therefore, there would be a difference only of degree between this process and that which results in the formation of the large "sausage-shaped" tumors sometimes found in the cord.

Others have explained the glia hyperplasia in other ways: Some have defined it as a reactive inflammatory process, others have held that the process has its beginning in the proliferation of glia tissue which takes place in tracts of the white matter where the nerve fibres are degenerated. Nonne's[2] case is a good example of this last—where a large gliomatous mass in the centre of the cord seemed for some distance to be in direct communication with the degenerated posterior columns of a tabetic cord.

The role of the bloodvessels in the production of, or at least in the later development of, syringomyelic cavities seems a very important one. Much has been written lately on traumatic syringomyelia (Kienböck, Brasch, and others) and the production in the cord of cavities by extensive hemorrhages, the result of trauma. The rule used to be laid down that these cavities were to be distinguished from syringomyelia, chiefly clinically, in that the symptoms of the former were sudden, and of the latter chronically progressive

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[1] Nord. Med. Ark., June, 1904.

[2] Archiv. f. Psychiatrie, Bd. xxiv.

(Van Gieson). Many claim, however, that the origin of a syringomyelic cavity may be in hemorrhages, at first small, later more extensive, therefore progressive. Spiller,[1] in 1896, reviewed the literature on this subject, and also spoke in favor of the probable origin of some syringomyelias in hemorrhages. These hemorrhages, he says, occupy usually the same portions of the gray matter of the cord chosen by syringomyelia, and often extend through a great length of the cord; as in the case of cerebral hemorrhages, they must become surrounded with somewhat thickened walls of neuroglia tissue. The reason that the gray matter is most frequently the seat of these small hemorrhages Spiller explains by the fact that it is much looser in structure, and more yielding than the white matter, the nerve fibres in it forming a reticulum, and not parallel columns, as in the white matter, and also because the gray matter is richer in vessels than the white. Kölpin,[2] reporting a case in 1905, says that he believes the cavity which he found, together with glia proliferation, has its origin in hemorrhages, at first very small, and gradually progressing; the effect of the first hemorrhages would be, by irritation of the surrounding tissue, to cause a reaction and proliferation of the same. As a result of this process, the bloodvessels themselves would be affected and a vicious cycle started. There was no history of trauma in this case, and the author believes the origin of the hemorrhages to have been either in the action of toxins or in a hemorrhagic diathesis on the part of the patient, and, from the fact that in his case the evidences of hemorrhages were seen in corresponding areas on both sides of the cord, he argues that perhaps certain sets of vessels have, in some persons, a predisposition to spontaneous hemorrhage. In Case II., above, the multiplication of the bloodvessels and the rarefaction of tissue appeared, it is true, in corresponding situations on both sides of the cord

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[1] International Med. Mag., 1896.

[2] Arch. f. Psych., 1905.

—namely, in the base of each posterior horn—and the cavity appeared to be growing only laterally and not anteroposteriorly.

Besides, by means of hemorrhages, the bloodvessels in the cord may cause destruction of tissue in other ways. Simple anæmia may cause rarefaction of tissue—ischæmic softening (Schlesinger). Or there may occur about the course of the bloodvessels a process of homogenization; later, liquefaction and absorption of the tissue, leaving cavities. This process of homogenization may be quite extensive, and perhaps accompanied by round-cell inflammation. Schwarz[1] reported such a case, and refused to call it a syringomyelia. Schlesinger remarks that this process occurring about the bloodvessels can hardly be held to account for the larger syringomyelic cavities. That stasis of the circulation, due to tumors or other cause, may give rise to cavity formation (Langhans) is a theory not generally accepted.

The importance of the connective tissue, which can be so often seen streaming off from the walls of the bloodvessels in the neighborhood of syringomyelic cavities, has been emphasized lately by Thomas and Hauser.[2] They found, as they show in a drawing, a cavity entirely lined by connective tissue, which latter seemed to be in direct continuity with the walls of a bloodvessel. The mechanism of the lesion, according to them, is: first, a multiplication of the bloodvessels with proliferation of their adventitia, and detachment of connective tissue from the same; these strands of growing connective tissue may isolate bits of nervous tissue, which will then disappear. Later there will be a disintegration of more substance, including the connective tissue itself, because of circulatory troubles. The proliferation of neuroglia they explain as a reaction against the invading strands of connective tissue. As to the nature of the original lesion

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[1] Zeitschr. f. klinische Med., 1898, Bd. xxxiv.

[2] Nouv. Icon. Salpêtrière, 17, 1902.

—the multiplication of the bloodvessels—they are not able to pronounce on it, but they say that its cause is without doubt inflammatory. In Case I., above, the largest cavity was found lined here and there, for a very limited extent, by connective-tissue lamellæ, the strands of which were in places seen to be in continuity with the walls of bloodvessels. There was also seen in the neighborhood of each of the cavities in this case a large number of small, thick-walled bloodvessels. The strands of connective tissue streaming off from the bloodvessel walls seemed, however, to penetrate to no very great distance into the surrounding tissue, and it is hard to see how they could have been the cause of anything else than a possible inflammatory reaction in the neighborhood. Schlesinger, in his monograph, shows a drawing in which nearly the whole of a syringomyelic cavity is lined by connective tissue. This connective tissue, he says, remains after the nerve substance has softened and disappeared, and forms the "protecting innermost layer of the tissue limiting the cavity."

Cavities in the cord seem oftentimes dependent on changes in the meninges (Cases of Japha, Oppenheim, and others). As Schlesinger puts it, one of four things must take place: (1) the changes in the cord are primary; those in the meninges secondary; (2) vice versa, the changes in the cord are secondary to those in the meninges; (3) both groups of changes are co-ordinated; (4) their occurrence together is merely a chance combination. Saxer's case of acute cerebrospinal meningitis, showing at the autopsy great glia proliferation all through the cord, and cavity formation, is given by Schlesinger as an example of the second of these explanations; and Wieting's explanation of "meningomyelitis chronica," in which chronic meningitis is shown to affect not only the dura but also the pia and the cord, is accepted. In support of the third of the above explanations, namely, that the changes in cord and meningitis are co-related, Philippe and

Oberthür[1] in 1899 reported a series of cases which seemed to show how syringomyelia and thickening of the membranes might be evolved together, under influence of the same cause, without being necessarily allied to one another. In their cases they found the greatest thickening and adhesions of the membranes (pachymeningitis symphysaire) at the level of the cervical swelling, and high up in the cervical regions, "regions most often showing the lesions of syringomyelia." In Case II., above, the thickening of the membranes was certainly most marked in the region of the cervical swelling, but here there was seen only a rarefaction of nervous tissue in the posterior part of the cord; the cavity was seen lower down in the cord, where there was distinctly less thickening of the membranes.

Nebelthau[2] has emphasized the importance of changes in the meninges, particularly syphilitic, in being the exciting cause in the production of cavities in the cord, and, speaking of syphilis, he remarks that in all cases of gliosis with cavity formation, in which the etiology is not clear, syphilis may be held responsible. By "not clear," he means not clearly due to congenital anomalies, to trauma with hemorrhage, or to inflammations of the meninges other than those syphilitic.

In Case II., above, where the cavity is comparatively small, it is easier to speculate on the possible origin and cause of the same than in Case I., where each cavity was fairly large. In Case II. the cavity was situated at a lower level in the cord than that at which the changes in the meninges were most marked; therefore the cavity was not solely dependent on these changes. The rarefaction of tissue and the presence of many small, thick-walled bloodvessels with round-cell infiltration at each lateral extremity of the cavity would point to some vascular disturbance as the cause here of the cavity formation, but anteriorly and posteriorly the cavity showed a wall lined by ependymal epithelium, with no rarefaction of neighboring tissue.

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[1] Revue Neurol., 1899.

[2] Deutsche Zeitschr. f. Nervenheilkunde, 1900, Bd. xvi.

This lining epithelium was apparently in continuity above, where there was no cavity, with a clump of epithelial cells separated from the central canal epithelium. Still higher, this clump of cells was seen to join the cells of the central canal. A possible explanation, in accord with Schlesinger's idea of the exciting cause of cell proliferation in such cases, would be that early in life, in the thoracic region of the cord, there existed, as the result of a congenital malformation, a nest of cells, with or without lumen, situated at some distance posterior to the central canal. Where these cells were farthest from the central canal, there the abnormal conditions in the cord must have been greatest, and the cord itself, therefore, most liable to suffer from pathological influence. When the thickening of the membranes and the vascular changes began the whole cord was affected, but though this thickening was greatest in the cervical region, the thoracic region of the cord, being congenitally the weakest, was most affected. The nest of cells there situated proceeded to proliferate. The vascular and meningeal changes, growing more marked, were then sufficient not only to arouse the process of proliferation in the abnormally situated cells, but also to cause those areas of markedly rarefied tissue which were seen in the posterior horns and in the posterior columns.

In Case I. all the cavities, excepting those in the anterior horns, had walls of thickened neuroglia, and none were lined by epithelium.

The opportunities for offering explanations of the origin of syringomyelic cavities are very abundant. Different authors have emphasized different points in regard to the etiology—the vascular changes, the changes in the meninges, the ependymal tendency to proliferate—and so forth, but each of these changes is generally recognized as having its own part to play, sometimes more, sometimes less important, in the production and extension of syringomyelic cavities.

The photographs are from sections stained by the Weigert hæmatoxylin method.

In conclusion, I wish to express my thanks to Dr. Spiller, under whose direction this work was done, for his kindness in giving me much help, and many suggestions.

THE SIGNIFICANCE OF JACKSONIAN EPILEPSY IN FOCAL DIAGNOSIS, WITH SOME DISCUSSION OF THE SITE AND NATURE OF THE LESIONS AND DISORDERS CAUSING THIS FORM OF SPASM.*

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WITH the exception of monoplegia or hemiplegia, probably no single symptom or symptom complex is so often made use of by the diagnostician in his efforts to fix the site of a tumor for the purpose of operation as Jacksonian epilepsy. This term is used here in a broad sense as meaning monospasm or hemispasm due to cortical or cortico-subcortical discharge, a spasm which usually exhibits, if closely studied, an initial symptom and a serial order of phenomena. As originally and as commonly employed, the mere use of the term is supposed to predicate the existence of a focal lesion of the motor cortex, and it is sometimes defined as a spasm limited to a single group of muscles, or at least to a few muscular groups. Irritation or instability of portions of the cerebral motor area, in by far the largest number of cases of Jacksonian spasm as here defined, is a direct or indirect cause of this form of epilepsy, but it is by no means true that it is always or even nearly always due to gross lesion of the motor zone. The greatest care should, therefore, be taken not to attribute to local spasm erroneous diagnostic value. While in many cases it is a most important guide, in others it may be misleading.

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* Read before the New York Academy of Medicine, April 19, 1906. From the Department of Neurology, University of Pennsylvania.

A distinction needs sometimes to be made between Jacksonian epilepsy and hemi-epilepsy. Some of the cases of hemi-epilepsy belong in the Jacksonian category, but they are not all to be relegated to it. The convulsive attacks following hemiplegia are frequently unilateral, but they may begin on the paralyzed side and become general. Occasionally they are confined to a portion of one side of the body. Frequently, probably usually, they are of cortical origin. When as is so often the case, the hemiplegia, especially in childhood and youth, is due to softening from thrombosis, the convolutional areas surrounding or in the neighborhood of the parts destroyed are often the seat of deep congestion and punctiform hemorrhages. These render the cortical centers unstable and convulsive discharges ensue, which may be confined to the limbs and face of one side, may be unilateral or may become general. The monospasm or unilateral convulsions which occur in cases of this description sometimes simulate very closely those which are due to brain tumor in the motor cortex or subcortex. They have the same mechanism as the Jacksonian spasms of tumor or other lesion, namely, irritation or instability of cortical motor cells. Hemi-epilepsy may, however, in some instances be due to an irritative lesion well removed from the cortex or to some toxic state.

It is scarcely necessary to present, as could easily be done, a large number of cases in which Jacksonian spasm has been an important or a deciding symptom in successfully fixing the site for operation in the case of a cerebral tumor. Many such cases have been recorded, and not a few by the writer. I shall refer only, and that briefly, to the histories of a few such cases.

Dr. C. H. Frazier, May 2, 1903, operated successfully for tumor of the motor region in a case under the care of Dr. Wm. G. Spiller,[1] which was seen by me in consultation. This case had the typical features of a neoplasm of the motor region, including Jacksonian spasm. The patient was a motor aphasic and agraphic with paresis of the right side of the face, right arm and right leg, exaggerated reflexes, and most of the general symptoms of brain tumor. Sensory symptoms were absent. He had had frequent attacks of spasm confined to the right side of the body, chiefly to the face and arm. According to the notes of the case, he always knew when these attacks of spasm were coming on by a twitching in the right eye for about five minutes before the spasms developed. The attack began by a peculiar sound in the throat, the eyeballs then rolled about, and the right arm and hand became implicated in the convulsive movements. Sometimes the right lower limb was affected, but never the left side. Consciousness was not lost, and the man did not fall into sleep after the convulsions. The attacks occurred usually when the man was awake, but several times when he was asleep. The convulsions lasted about three minutes, sometimes longer.

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[1] Spiller, Wm. G.: A Report of Five Cases of Tumor of the Brain, with Operation. The American Journal of Medical Sciences, February, 1904.

I have had under observation a number of cases of cyst of the motor region in some of which unilateral convulsions or monospasms have been present, these being evidently due to the unstable condition of the cortex surrounding the cyst. In several such cases Dr. C. H. Frazier has operated, exposing the cyst, and in some instances cutting away its roof with some benefit in the direction of lowering the frequency and severity of the attacks.

A tumor largely limited to the motor region was removed by Dr. W. W. Keen from a patient seen by me in consultation with Dr. M. Behrend and Dr. Keen. This patient's symptoms began with weakness in the left upper extremity about eight months before the operation. She had an attack of vertigo with monospasm about six or seven weeks before operation. She gradually developed almost complete paralysis of the left upper extremity. She had absolutely no power in the limb below the elbow. The left lower extremity was also almost completely paralyzed. The knee jerk and the deep reflexes on the left side were all exaggerated. Ankle clonus was persistent. The same reflexes on the other side were prompt, but not abnormal. The Babinski response was present on the left; on the right the plantar response was prompt, but not abnormal. Examination made for sensation showed no sensory loss or impairment.

The Jacksonian spasm from which this patient suffered was observed in one attack by a trained nurse who reported that the spasm began in the arm by a lifting movement of the entire limb, and quickly extended to the hand and leg, and sometimes to the left side of the face. The operation was osteoplastic and was so planned that the opening included an exposure of the brain two thirds in front and one third behind the central fissure. A large sarcomatous tumor was successfully removed, the patient making a good recovery. This case, which I give only in its broad outlines, although reported to one or two societies, has not been hitherto published.

In one case of tumor of the motor region seen by me in consultation with Dr. T. C. Potter and Dr. Wharton Sinkler, the growth after accurate localization, was removed by Dr. Wm. J. Taylor.

The patient had no objective sensory symptoms although she at times complained of a numb feeling in the left side of the face, left arm and left leg, which were paretic, the loss of power being marked in the lower extremity. She had several convulsive seizures, chiefly affecting the limbs of the partially paralyzed half of the body. All the deep reflexes on the left side were increased, persistent ankle clonus being present. The Babinski phenomenon was elicited on the left side. Headache was not conspicuous, but vomiting occurred, and double optic neuritis was present.

One of the most interesting of my motor region cases was that in which successful operation was performed by Dr. W. J. Hearn at the Philadelphia Hospital, Oct. 21, 1902. This was a case of localized pachymeningitis and gumma of the cortex. The patient was a man twenty-seven years old, who had been struck in the left parietal region eleven years previously and who also probably had syphilis. Vertigo, headache, vomiting and optic neuritis were all marked symptoms during the development and progress of the case. His chief symptoms just before the operation were, extreme pain in the left parietal region, frequent spasmodic seizures beginning in the right hand and arm, and later involving the right leg and right side of the face, with temporary loss of power in the right arm and leg after the seizure. Tenderness was extreme over the left parietal scar and over the surrounding region for a distance of three inches or more. Grip in the right hand was slightly impaired. Sometimes immediately after a spasm loss of power in the right arm and forearm was marked. Biceps jerk in both arms was slightly increased, but triceps jerk was normal in both. Tactile, pain, and thermal senses and stereognostic conception were normal. Voluntary and resisted movements in the right leg and foot were slightly impaired. Knee jerks were exaggerated on both sides, more markedly on the right. Ankle clonus was present on both sides. The Babinski response could not be obtained on either side.

In connection with these cases on which operation was performed, and several of which have been published in detail, I am able through the courtesy of Dr. C. W. Burr and Dr. R. L. Lavenson, to present the notes of an unusual case of Jacksonian epilepsy due to small cortical lesion. It will be seen that this patient had for a long time a continuing clonic spasm of the upper extremity, preceded by a marked Jacksonian spasm with unconsciousness. For a time the clonic spasm was intermittent. A few similar cases have been reported under the title of epilepsia continuans. This interesting case will later be published in full. The following are notes furnished by Dr. Burr:

" The patient was a woman, fifty-five years of age, who, four years after amputation of the breast for carcinoma, began to have convulsive twitchings in the left arm. At first the spasm continued for a few minutes to a half hour and would recur from ten to twenty times a day. About a week later quite violent convulsive movements commenced and were persistent. They decreased but did not cease during sleep. There were flexion and extension of the fingers, and flexion and pronation of the forearm. If the muscles were tapped the spasm increased greatly. Voluntary movement in the arm also increased the spasm. There was only slight loss of power. The patient did not complain of the left leg at all. On examination, however, there was seen a coarse, persistent, clonic, spasmodic tremor. She was able to walk and there was no palsy of the leg.

The left knee jerk was increased, the right normal. There was no ankle clonus, no Babinski sign. She had no headache or vomiting. Once, before the spasm became persistent, the patient had a localized fit with unconsciousness, following which there was loss of power and sensation in the arm for a few minutes. This was the only time in which the sensation or consciousness was affected.

"The brain showed the following: The whole organ was somewhat atrophied. In the ascending frontal convolution of the right side, at about the hand center, was a spheroidal tumor about one third of an inch in diameter, arising from the pia and deeply compressing the cortex so that the tumor was buried in a socket. On microscopic examination it proved to be a carcinoma. There was another small tumor about the same size in the pia on the inferior surface of the left lobe of the cerebellum. I do not think that the latter had anything to do with the symptoms."

These cases have been given, omitting many details, to show the importance of Jacksonian spasm in gross lesions of the cortex, especially in tumor and localized meningitis. In such cases a few features would seem to point with comparative certainty to gross lesion. In these and in a large number of similar cases, other evidences of gross lesion in addition to the spasm were present. They were chiefly monoplegia or hemiparesis with exaggerated reflexes on the side of the body in which the localized spasm occurred. In most cases close investigation of the conditions of motility and of the reflexes shows this persisting impairment of power and abnormality of reflexes.

So important is this question of Jacksonian epilepsy in the focal diagnosis of brain tumors, that it will be advisable to consider at length the seat and nature of various lesions and conditions producing this symptom. The diagnostician must bear in mind (1) that tumors situated in other parts of the brain than the motor cortex may cause Jacksonian epilepsy; (2) that other lesions besides tumors situated in the motor cortex may cause this form of spasm; (3) that it may occur in toxic and other disease in which no demonstrable focal lesions are present; (4) that a spasm closely counterparting the Jacksonian type may be observed as a reflex or a hysterical disorder; and (5) that Jacksonian epilepsy may be simply an integral part or the entire expression of a case of idiopathic epilepsy.

Jacksonian epilepsy due to cortical discharge may occur as the result of tumors in other parts of the brain than the motor zone. The irritation owing to its intensity, spreads to the motor cortex. It is probable also that in some of these cases unusual instability of the motor cells plays some part. Cases of this kind, in which the lesion is in the cerebrum proper, and absolutely outside of the motor zone are comparatively rare, and the focal diagnosis can only be made in them by a full consideration of the non-motor symptomatology.

These cases in which Jacksonian epilepsy is a so-called distant symptom are not to be confounded with the comparatively common cases in which the spasm is an invasion symptom. A midfrontal or even a prefrontal tumor not infrequently invades caudally until the motor region is grazed or implicated, Jacksonian spasm of typical form showing itself after psychic, graphic and speech disorders have become prominent. In these cases, if the tumor be meningocortical in position, spasm may precede paresis or paralysis, although sooner or later, in accordance with the amount of pressure or destruction of the motor region, the patient will become monoplegic or hemiplegic. Tumors invading backwards from the prefrontal towards the motor region are, in my experience, much more likely to cause Jacksonian epilepsy than those which originate in the parietal lobe and advance forwards. I have recorded several cases of parietal tumor in which the motor cortex and subcortex were invaded and in which Jacksonian epilepsy was not present even at a late stage of the disease. Destruction of the sensory cortex and subcortex, before involvement of the motor projection fibers or cortex, would seem to give a certain immunity from such spasm. A case beginning and continuing for some time with such symptoms as cutaneous hypesthesia, ataxia of the upper extremity and loss or impairment of the muscular sense and of stereognostic conception, frequently has paresis and then profound paralysis among its later or terminal symptoms, but this is not often accompanied by Jacksonian spasm.

In some instances, Jacksonian epilepsy, apparently of the usual motor area type, is observed in tumors of the cerebellopontile angle or of the cerebellum. In two cases which have come under the writer's observation, cerebellopontile tumors caused Jacksonian epilepsy or what appeared to be this affection. The first of these cases has been reported in detail by Dr. T. H. Weisenburg.[2] The tumor sprang from the eighth nerve, and the chief focal symptoms were one-sided deafness, tinnitus, facial monospasm, hypesthesia of one side of the face, nystagmoid movements, slight paresis of the right abducens, and vasomotor and cardiac disturbances. Severe headache, nausea, vomiting and optic neuritis were also present. I examined this patient many times and on several occasions witnessed the facial monospasm which usually showed the same features. In this spasm the mouth was drawn as far as possible to the left and the eyelids were brought together. Only the left side of the face was involved in the seizure. On one occasion it was thought that the facial spasm was accompanied by some spasmodic movements of the left hand, but this was doubtful, and even the observer thought it may have been a voluntary movement.

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[2] Weisenburg, T. H.: New York Medical Journal and Philadelphia Medical Journal Feb. 11 and Feb. 18, 1905. This paper is one of a series of contributions by Drs. Mills, Frazier, de Schweinitz, Weisenburg and Lodholz, published in the above numbers of the New York Medical Journal and reprinted in a monograph on Tumors of the Cerebellum.

In a second case seen by me recently,[3] one in which a tumor was successfully removed by Dr. John H. Gibbon from the cerebellopontile recess, Jacksonian epilepsy, or at least facial or faciobrachial spasm, was one of the most striking features in the symptom complex. In this case the general symptoms of brain tumor, including headache, vertigo, nausea and vomiting, and optic neuritis with atrophy were present, the chief focal symptoms being deafness in one ear, and on the same side facial and abducens paresis with nystagmoid movements and the spasm above described. In the convulsive seizure the face was markedly drawn to the left, and the spasm in some instances passed to the arm and hand of the same side. Spasmodic attacks occurred at intervals for several months. In at least one or two of them observed before the operation tonic spasm involved both sides of the body.

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[3] This case will later be published in detail by Dr. John H. Gibbon and the writer.

Some discussion was had as to whether this case could be a prefrontal one invading the face and arm areas, or, as it proved to be, a cerebellopontile growth. The diagnosis was not as easy as might at first sight appear, as abducens paralysis occurs in tumors variously situated; and at first there was some question as to the central origin of the deafness. Decision was finally given, however, in favor of the cerebellopontile angle, and the operation was the most successful which has as yet been performed for a tumor situated in this locality.

Collier[4] calls attention to two cases in which a tumor of the cerebellum caused Jacksonian attacks. Local convulsion of slow spread and confined to the arm and face was repeatedly observed. Some bilateral spasticity was present in these cases. No lesions except the cerebellar tumors were present, but the ventricles were considerably distended. Hemi-epilepsy is noted by Collier as having occurred in one case of tumor of the pons and in one case of tumor of the cerebellum. General convulsions occurred in other cases. Collier is inclined to attribute the local spasms, in some cases at least, to hydrocephalus. He warns against confounding Jacksonian spasms of ordinary type with Hughlings Jackson's lower level fits. Grainger Stewart and Gordon Holmes[5] direct attention to this point in the same number of *Brain* as that in which the article by Collier appears.

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[4] Collier, James: The False Localizing Signs of Intracranial Tumor. Brain, part iv, 1904.

In considering Jacksonian epilepsy in its relations to the focal diagnosis of a cerebral tumor, the question of spasm due to dural irritation must not be overlooked, especially as certain forms of neoplasm commonly grow from the inner surface of the dura. Convulsion, local or general, due to intense dural irritation may occur as one of the symptoms of a brain tumor situated anywhere within the cranial cavity as has been demonstrated by clinicopathological observation corroborated by physiological experiment. Galvanization or faradization of the dura sometimes causes intense and generalized convulsive attacks. On two occasions I have seen such spasms produced by faradic applications to the dura while tests were being made during operation. The experimental investigations in this field are well known. One point of distinction between cortical Jacksonian spasm and the convulsive affection due to dural irritation is that in the latter the spasm, if observed at the start, will be found usually to begin in the face or limbs of the side of the irritation, spreading however rapidly to both sides of the body; so rapidly that the initial spasmodic phenomena may pass unobserved. The spasms almost always become generalized, and tonic spasticity is prominent. Such spasms are really reflex in character, the irritation being transmitted to the bulbar centers of the same side, and thence to those of the opposite side. In addition to the peculiarities of the spasm just noted, the diagnosis from cortical Jacksonian epilepsy will, of course, have to be made by the other focal symptoms present, as by those indicating parietal, occipital, temporal or midfrontal disease.

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[5] Stewart, T., Grainger and Holmes, Gordon: Symptomatology of Cerebellar Tumors, A Study of Forty Cases. Brain, vol. xxvii, 1904.

In some cases in which tumors grow from the agglutinated dura and pia in the motor region, a confusing picture of more or less generalized convulsions is presented. I have seen such cases in which it was possible, especially with after-knowledge, to pick out that part of the spasmodic outbreak which was due to irritation of the motor cortex, and that which was dependent on dural irritation.

The nature of the lesion of the motor area causing localized spasm next requires attention. That other lesions situated in the motor cortex besides tumors may cause Jacksonian epilepsy is, of course, well understood, but the fact is not always given as full consideration as its importance demands when the question of operation is under discussion. Decision as to the nature of the irritative lesion is particularly difficult in those cases, not inconsiderable in number, in which such general symptoms of brain tumor as persistent headache and optic neuritis are absent. I have known both of these to be absent in tumors of considerable size, while vertigo and nausea and vomiting have been so little marked as not to direct decided attention to their probable cerebral origin.

The gross lesions of the motor area which cause monospasm or hemispasm, counterparting that produced by neoplasm, are, (1) depressed fractures; (2) localized meningitis; (3) meningeal or cortical hemorrhage; (4) focal hemorrhagic encephalitis or cortical polio-encephalitis; (5) focal necrosis occurring from embolism or thrombosis, including cases associated with generalized arteriosclerosis.

It would, of course, only be in the case of an old fracture of the inner table with slight or no outward evidences of the traumatism, that the mistake would be likely to be made of holding that Jacksonian spasm was due to a tumor. It occasionally happens that an injury to the head which leaves only a small scar in its wake results after a considerable time in the development of Jacksonian spasm. Uusally in such cases both headache and vertigo, more or less severe and persisting, are present and a tumor may at first be inferred. Close investigation into the history of the case and as to evidences of injury will usually clear up the diagnosis. A mistake is not necessarily of serious import in such a case, as tumors sometimes develop at the site of old injuries, and in any case operation is indicated for the relief of the lesion causing the irritation. Localized meningitis with adhesions or even an abscess may, of course, occur at the site of such a traumatism.

With regard to localized meningitis not of traumatic origin, and not connected with neoplasm, this is usually of syphilitic origin. The case referred to in which Dr. Hearn operated successfully was one of localized pachymeningitis, or rather of conjoint gummatous inflammation, of the dura and pia arachnoid with exudate of sufficient extent and consistence to constitute a tumor mass. It is in this form of gummatous meningitis, more or less amenable to active specific treatment with mercury and the iodides, that too hasty decision in favor of operation is occasionally gven. On the other hand, operation is undoubtedly indicated in some cases with a syphilitic history and with clear evidences of vascular and meningeal specific disease, but in which the case does not respond even to the most energetic medicinal treatment. In some of these cases occlusion of vessels takes place and deposits become organized so that they cannot be influenced by absorbent remedies, and the lesion, although at first simply an active localized meningitis, becomes an inert and irritating mass which should be dealt with by the surgeon.

Little needs to be said about Jacksonian spasm due to meningeal or cortical hemorrhage in connection with our study of the focal diagnosis of brain tumors. Supradural and subdural hemorrhages are so constantly due to traumatisms, and their symptoms so frequently acute in onset, that the spasm which is sometimes present in the subdural variety, and may be largely confined to one half of the body, is not likely to be confounded with the spasm of a brain tumor. The diagnosis will be decided by the history of the case and by the well-known phenomena of dural hemorrhage, such as contralateral paralysis, dilated pupil on the side of the lesion, varying conditions of consciousness, and peculiar changes in pulse, temperature and respiration.

Cortical hemorrhage of slight depth and irregularly distributed sometimes occurs in cases of sinus and venous thrombosis. Horsley[6] has reported a case of this sort in which Jacksonian spasm clearly defining certain subareas of the motor zone was the most characteristic symptom. In such a case the history of acute or subacute onset, and the symptoms of sinus and venous thrombosis would guide. A hemorrhage or a cyst remaining after a hemorrhage from one of the branches of the medicerebral artery may give rise to Jacksonian epilepsy and other symptoms simulating somewhat those of a tumor. The diagnostician is usually not called upon to differentiate in such a case until weeks, months or years have elapsed after the original lesion, and as the history is sometimes imperfect or confusing, and as the patients who suffer from such attacks are usually victims of arteriosclerosis, they may have some headache and vertigo, which may be regarded as general symptoms of a neoplasm. Associated with the paresis or paralysis commonly present in such cases, may be recurring Jacksonian seizures. Close study of the clinical phenomena will, as a rule, be sufficient to throw the weight of opinion in such cases against tumor or localized meningitis.

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[6] Horsley, Victor: *Ibid.*, April, 1888.

A rare form of cortical disease which causes Jacksonian epilepsy and may be regarded as a tumor of the motor zone in an early stage of its development is focal hemorrhagic encephalitis. Such cases are now and then observed, both in children and in adults. Recently a case of this kind died in my wards in the Philadelphia General Hospital. The patient was an aged woman, who had a number of carefully observed Jacksonian spasms involving the arm and face. Consciousness was retained in the convulsive attacks. The different parts of the arm and face were usually involved in about the same order. The hand, for instance, was drawn somewhat to the left, and the angle of the mouth was drawn outwards and upwards; the eye was closed and the brow wrinkled, but without any frowning movement or movements of the eyeballs or jaws. Dilatation of the left nostril and movement of the platysma took place. In the spasm the movement was confined to the deep extensors of the fingers and thumb and group of ulnar extensors, there being no movement of the upper arm and shoulder muscles. Examination showed that the patient was distinctly paretic in the left side of the face and arm, but not in the leg.

She had no affection of sensibility and the reflexes were not altered to any marked extent. She was probably hemianopsic, although this was not clearly determined owing to the mental condition of the patient.

The necropsy showed many interesting pathological conditions in various parts of the body, including a circumscribed area of hemorrhagic encephalitis in the motor cortex, this evidently having been the cause of the Jacksonian spasm.[7]

One of the forms of focal cerebral disease which must be taken into full consideration when the question of operation, and especially for operation for brain tumor in the motor region, is presented is that of arteriosclerosis which has caused either local instability or local softening with symptoms which simulate those which are exhibited by neoplasms. The cases of this kind most difficult of decision are those in which Jacksonian epilepsy is present. In such a case the history will be that of gradual cerebral failure dependent usually upon generalized arteriosclerosis with its renal and cardiac accompaniments, and a record of seizures and of progressive loss of power of the limbs, or the limbs and face of one side. Instead of the motor manifestations, or in addition to them, the symptoms may be loss or impairment of different forms of sensibility or of stereognostic conception or some disturbance of speech and writing, according to the parts involved outside of the motor region. The patient may have an attack of transient dazing or of vertigo at one time, and at another, with this or independently of it, some form of localized tonic or clonic spasm; the face may twitch on one side or the fingers and hand may be clinched, or the arm may lift or the toe may be extended. Still later this illy defined Jacksonian attack may merge into a facial, brachial, brachio-facial or hemispasm. In brief, the record is one of Jacksonian attacks slowly and irregularly developed to their full stature, usually with late coming on one-sided paresis or paralysis.

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[7] This case will be reported in full in a paper which will be presented at the meeting of the American Neurological Association, to be held in Boston, June 4 and 5 1906.

Such a case is to be differentiated from one of brain tumor by a close study of the manner in which the symptomatology has developed, by the exclusion of the general symptoms of brain tumor and by taking into consideration the evidences of renal, cardiac and general arterial disease. Optic neuritis is practically always absent, and a very skillful ophthalmoscopic examination will often show evidences of sclerotic disease of the vessels and of some grade of optic nerve atrophy. Headache if present is not that which is often characteristic of brain tumor. Nausea and vomiting are generally absent.

Examination of the urine may show albumin or casts or both. Dilatation with hypertrophy of the heart is present, and arterial tension is nearly always high. The possibility of the coexistence of arteriosclerosis and brain tumor must not be overlooked.

As in this connection I am chiefly concerned with the discussion of Jacksonian epilepsy, the interesting general subject of arteriosclerosis and focal cerebral lesions will not here be discussed in detail. I have had under observation several cases of necrotic lesion in which the diagnosis of brain tumor was made at some period in the history of the case, and in which operation was performed.

In one of my cases in which the localizing symptoms were astereognosis, diminution in pain and temperature senses, word deafness and word blindness, amnesic aphasia, paraphasia, lateral homonymous hemianopsia and late hemiparesis, operation was performed and revealed an area of necrotic tissue. A study of the history of this case shows how mistakes may be made with regard to what appear to be the general symptoms of brain tumor. At one period a most skillful ophthalmologist reported in effect that while optic neuritis was absent, the conditions present might indicate that it was imminent. Demonstrable neuritis was never present, neither before nor during the several years which intervened before death and after the operation. The patient complained at times of headache, but this was not severe. His first marked symptoms were initiated with an attack of dizziness and numbness, but this dizziness proved not to be the vertigo of brain tumor, but that which is associated with arteriosclerosis and spreading vascular lesions. Nausea and vomiting were not present.

In another case which I saw in consultation with Dr. J. H. W. Rhein, and in which an osteoplastic operation was performed by Dr. A. C. Wood, an area, probably of necrosis, was found situated largely in the motor zone. The patient was a woman fifty-seven years old, whose train of serious symptoms was ushered in by a convulsion. Later she developed recurring aphasic attacks preceded by incoherence, excitement and fright; still later paralysis of the arm and leg and paresis of the face appeared. The reflexes were increased on the paralyzed side, including slight ankle clonus and the Babinski response. Sensation was everywhere preserved. Ophthalmoscopic examination showed moderate pressure signs where the arteries crossed the veins; otherwise the eye grounds were entirely normal. Some albumin was found in the urine, and at one examination one granular cast.

While true Jacksonian epilepsy was not a manifestation in either of these cases, they belong to the class of cases in which vascular lesions give a symptomatology somewhat closely simulating that of brain tumor. Cases with similar general symptoms and Jacksonian epilepsy are occasionally regarded as tumors of the motor region.

The diagnostician must not lose sight of another class of cases in which vascular lesions occur with brain tumor, but in other locations than that of the tumor.

The occurrence of hemorrhage or necrosis due to thrombosis or embolism, and especially the former, with brain tumor may, as Collier points out, give false localizing signs. He records one case in which operation revealed an area of necrosis which had evidently caused the localizing symptoms which guided in the choice of a site for operation, and in which subsequent necropsy showed the presence in addition of a large tumor. Hemorrhage is not very uncommon in the progress of a case of brain tumor, and may be in, around or at a distance from the site of the growth. The concurrence of vascular lesions with neoplasms is what might be expected when it is considered that tumors are so frequently observed after middle life, and in those who exhibit senile or presenile arteriosclerosis.

Hemi-epilepsy, or Jacksonian epilepsy of smaller range, is sometimes observed as a toxic or diathetic affection. One of the most striking cases of Jacksonian spasm ever seen by me was in a patient suffering from diabetes who had an attack of spasm while consulting me in my office. He remained conscious during the attack and even succeeded in talking a little about his feelings, although he did this with difficulty. The arm and face were the parts affected, the spasm beginning in the distal portion of the limb and extending to the other parts. It is now well known that monoplegias and hemiplegias sometimes occur in the course of nephritis, and in some of these cases necropsy has shown that all forms of gross lesion were absent. A few such cases have fallen under my own observation, necropsy showing no gross lesion. In other cases, however, where the toxic form of uremic monoplegia or hemiplegia was diagnosticated, necropsy has shown a hemorrhagic or embolic lesion or local cerebral edema. Uremic convulsions occasionally are of Jacksonian type, and more frequently begin in the limbs or face of one side, the attack later becoming general. Jacksonian epilepsy is also sometimes observed in Korsakoff's disease or as an acute alcoholic manifestation, and may indeed be present in any form of toxic or infectious disease. These toxic spasms need only passing reference in the discussion of tumors of the cerebral motor zone. It is only in extremely rare cases that the diagnosis is not evident, — cases in which the vertigo and headache are present, or in which an optic neuritis of toxic origin is regarded as due to cerebral neoplasm. The diagnosis is, of course, to be made by a study of the history of the case, by examinations of the urine and blood and by the absence of the typical general and focal symptoms of a brain tumor.

The dural epilepsies which have been considered on the previous pages are reflex epilepsies; in fact, they probably afford the best possible illustration of a severe convulsive attack due clearly to reflex causes. A tumor situated in the dura, or a galvanic or faradic current applied to this membrane, stimulates the sensory branches of the fifth nerve distributed towards its inner surface, and this excitation conveyed to the bulb produces the severe and characteristic spasmodic symptoms to which reference has been made. The broad assertion may be made that peripheral irritation almost anywhere in the body may cause, in rare cases, a convulsive attack, and that this may in still rarer instances assume the Jacksonian type. In such a case, as in the toxemic convulsive disorders, the spasm indicates cortical discharge, and it is probable that the occurrence of the attacks is conditioned by unusual inherited or acquired instability of the motor cortex. It is only in a very unusual case that the diagnosis of brain tumor would be considered in such reflex spasms. Some years ago I reported a case of this kind in which the spasm, typically Jacksonian in its initiation and spread, was due to a fibroma of the palmar surface of the hand. The attacks became frequent and severe. They continued for some time after the operation for removal of the palmar growth, but eventually disappeared entirely. The patient was of a neurotic constitution and the intense peripheral irritation in this case undoubtedly affected the unstable cortex. The history of the case and the discovery of the source of peripheral irritation are sufficient to guide the diagnostician.

Some hystero-epileptic attacks bear a close resemblage to Jacksonian epilepsy. The presence of hysterical stigmata and the absence of the symptoms, general and focal, indicative of organic brain disease are sufficient to prevent error in diagnosis.

The question of the differential diagnosis of Jacksonian epilepsy due to tumor or other gross, and possibly operable, lesions of the motor zone, from idiopathic epilepsy is one of great moment. Collier goes so far as to say that the commonest cause of Jacksonian spasm is idiopathic epilepsy. In several cases in recent years I have known operations to be done without revealing any tangible lesion, the diagnosis of the nature and site of the lesion having been chiefly made because of the characteristics exhibited by the Jacksonian spasm which was the chief feature of the symptomatology of the case. In some of these cases a small undiscoverable growth was present in the subcortex or just outside of the limits of the cerebral surface exposed, this being demonstrated by subsequent developments of a second operation or by necropsy. In other cases observation of the patient over a considerable time, as well as a more thorough consideration of all the features of the symptomatology of the case, have made it clear that the Jacksonian spasm, although apparently typical, was in reality simply an integral part of the attack of true idiopathic epilepsy. In rare instances the idiopathic case shows almost restricted Jacksonian manifestations, at least the spasm continues to be largely a hemi-epilepsy. It is extremely rare in an idiopathic case for the spasm to remain limited to one limb, or to one side of the face, although this is not unknown and patients, as is well known, may have abortive attacks of epilepsy with a sensory or sensorimotor manifestation of a very transient character in a part to which the aura of a completed attack is commonly referred. In some cases in which Jacksonian spasm occurs as a part of the manifestation of an idiopathic epileptic attack, the Jacksonian spasm occurs, so to speak, inside the general convulsion; in other words, the patient during the seizure has clonic and tonic spasm which involves more or less irregularly all parts of the body, but in which the spasm shows itself most pronouncedly in the limbs or in the face and limb or limbs of one side of the body.

Every neurologist has had frequent occasion to confirm the truth of Dr. Hughlings Jackson's well-known opinion that almost every case of idiopathic epilepsy, if studied carefully enough, will be found to have had local spasm as the initiating phenomenon of the general attack. It is true that it is almost impossible in many cases to get a clear record of the initial local spasmodic phenomenon by questioning those who observe the attacks, and even the trained physician or nurse may fail to make this observation largely because of the speedy manner in which the spasm radiates to many parts of the musculature.

In a case recently observed, a convulsive attack began with a somewhat suffused appearance of the face, the head and eyes turning to the left conjointly with a spasmodic movement not very pronounced in the right upper extremity, chiefly affecting the forearm, hand and fingers, which were held in a partially flexed position. The spasm did not, in the attack observed, become entirely generalized, but the left lower extremity was spasmodically extended. It was reported to me that in other seizures the spasms were or became general. In a second case spasm began with a movement performed by the pterygoid and masseter muscles of one side, and facial twitching, the patient becoming unconscious, with generalized tonic spasticity. In a third case the spasm, which soon became general, was in its earliest stage confined to tonic spasticity of the extremities of one side. An opinion in favor of operation was refused in these cases, the evidence pointing to idiopathic epilepsy. With the exception of the localized spasms, other signs of a growth or of localized meningitis were absent. These cases could be readily multiplied, and probably more striking illustrations of initial Jacksonian spasm in cases of idiopathic epilepsy, furnished. I refer to them simply because of their recently having come under notice and because they were under observation for the purpose of a decision with regard to surgical procedure. Among the operative cases of which I have notes are two in which osteoplastic operations were made by Dr. C. H. Frazier, and to which brief reference might be made. In the first of these cases it is probable that the true solution of the case is that it is one of the idiopathic epilepsy very closely simulating a case of brain tumor with Jacksonian signs. In the other it is probable that sooner or later a subcortical neoplasm, at present small, will appear on the surface or in some position close to the limits of the exposed portion of the cerebrum.

The first of these cases was a man thirty-four years old, many of whose spasmodic seizures were carefully studied in the hospital of the University of Pennsylvania. These attacks usually began with a frequently repeated trisyllabic sound, the patient's head was turned somewhat to the right, his face drawn on this side and the right arm flexed at the elbow. During four or five months before the patient came under observation, he had had a series of attacks, about half a dozen in all, some of which were mild and others severe. The patient knew when the attacks were coming on by a feeling of confusion and dizziness and by a movement of the right forearm, which flexed and extended on the arm. He remained conscious long enough to experience and observe these phenomena. The light attacks passed off without the spasm becoming general. He had on one or two occasions what appeared to be the epileptic status, or at least a series of spasmodic attacks extending over three or four hours, and after one of these periods he remained unconscious for two days. In the severe and recurring attacks the spasm became general, although almost invariably beginning in the same way and always being more marked on the right side of the body.

Examination for all forms of sensation was negative; the grip of the right hand was not quite as good as that of the left, but no other evidences of paresis of the limb were present. Voluntary movement of the right side of the face was weaker than that of the left. The right knee jerk was exaggerated, but ankle clonus and the Babinski reflex were absent. Optic neuritis was also absent, and the patient did not suffer much from headache.

Operation was performed in this case, an osteoplastic flap being made over the midfrontal and the lower two thirds of the motor region. No lesion was discovered except that the dura seemed unusually thick and dense. This patient had a specific history, and the question of a gumma or gummatous meningitis was considered, but in the light of the operation and with the history which has only been briefly sketched, the case may have been one of idiopathic epilepsy. Of course it is possible that a subcortical prefrontal growth developing backwards may later be revealed.

The second case was that of a man thirty-two years old, of healthy appearance, without any history of specific disease or alcoholism. He said that he had been perfectly well until four weeks before he came to the hospital, to which he was referred by Dr. M. H. Fussell under whose care he had been as a private patient. Some three months before his admission he had given himself a sudden strain in attempting to get on a moving car, but otherwise had no history of injury. Four weeks before admission he noticed for the first time feelings of numbness in the thumb and finger of the left hand immediately followed by twitchings of of the left eyelid. Later he began to have frequent attacks of localized spasm which affected the left upper extremity alone, the left side of the face alone and both the left side of the face and the left upper limb. These were observed by various members of the neurological staff. The patient kept an account of his attacks which became very frequent before the operation. He remained conscious in them and described the peculiar sensations felt by him and would often detail the manner in which the attack was progressing, saying, for instance, " Now it is in the fingers, now in the arm, now in the eyes, now in the face," etc. The spasm was distinctly Jacksonian, being either brachial, facial or faciobrachial in form. In one of his seizures the patient first complained of numbness of the thumb and index finger of his left hand. In a few seconds a distinct twitching of the left side of the occipito-frontal muscle was noticed, followed quickly by movements of the orbicular and levator palpebræ, and next by movements of the left side of the mouth and face. Finally the right occipito frontalis took part in the spasm. The patient remained conscious, and in this spasm no part but the face was affected.

In another seizure first a fine clonic spasm of the thumb and forefinger was observed, quickly followed by supination of the hand and forearm, flexion of the hand on the forearm, then by biceps, anconeus and deltoid spasm. When the spasm began to affect the flexors of the forearm, the left occipito-frontal and the left side of the face become involved also after the manner described in the previous notes. At the close of the main attack movement of the subscapular muscle could be distinctly felt, although not observed during the main attack. No movement of the eyeballs was observed. At no time during the attack was the patient unconscious. Many other attacks were observed, these recurring with great frequency, even as often as twelve or thirteen times in one day. In all either facial or brachial spasm or both were present, the details of the twitching differing considerably as regards the movement of the musculature involved, especially in the upper extremity. I had the opportunity of showing a portion of one of these attacks at one of my clinical lectures, the patient walking into the arena during the seizure. The occipitofrontal, orbicular and facial movements were still evident as he entered. The spasm in the arm was passing off, but the hands still slowly flexed and extended, the forearm being held semi-flexed on the arm.

Examination showed some slight general weakness of the left upper extremity, and in the thumb and fingers marked impairment of power. Weakness was marked in pronation and supination and in flexion and extension of the hand and fingers. Extension of the second and third phalanges and flexion of the first phalanx of the index finger were almost lost. He was unable to oppose the thumb to any of the fingers or to abduct the thumb. The musculature of the left arm was distinctly flabby as compared with that of the right, and slight wasting of the muscles of the left forearm was evident. Movements of the fingers and hand were awkward, and attempts to make the finger to nose test caused some coarse tremor of the hand. The muscles affected by the spasm in the left side of the face, like those of the upper extremity, showed some impairment of power, but this was not marked. The deep reflexes in the left upper extremity were exaggerated.

Other parts of the body than the left side of the face and left upper limb showed no abnormal conditions although the knee jerks and deep reflexes generally were rather over-prompt. No loss of cutaneous sensation was anywhere present, and the patient was not astereognostic. Hemianopsia was absent, as were also all disorders of ocular movements. Repeated examinations of the fundus showed no optic neuritis, although some blurring of the disk was present. The patient complained at times of dull headache, but pain in the head was not that characteristic of a brain tumor. It was decided, however, that the symptoms probably pointed to a small growth in the cortex or subcortex of the arm and face areas.

Dr. Frazier performed an osteoplastic operation, uncovering the arm and face areas in the usual manner. No tumor was found. At one spot the coloration of the surface seemed somewhat lighter in appearance but incision here revealed nothing, and it was decided not to explore the subcortex any further. The patient made a good surgical recovery. He had a few left-sided spasms during a day or two after the operation, but these ceased, and at the time of writing, between six and seven weeks after the operation, he had had no recurrence of them. The arm is, however, considerably more paretic, but it has improved in this respect since the first few days.

This case seemed to present in almost every respect the characteristics of a small gross lesion, and it is not improbable that a subcortical tumor will eventually make its appearance on the surface. The case is similar to one which came under my notice at the Philadelphia General Hospital in the service of my colleague, Dr. Wm. G. Spiller, and which will later be published in full by Dr. Spiller and Dr. Edward Martin who operated. This patient was a man sixty years of age who had had convulsions confined to the left side for eight years. The spasm implicated the left upper and lower limbs and left side of the face, and the face and eyeballs were drawn to the left. The attacks had become more severe during the past four or five years, and five were observed by Dr. Spiller within an hour. An operation was performed and a peculiar appearing area was uncovered, but it was not distinctly pathological. The patient died two days after the operation. A very small sarcoma was found in the right second frontal convolution, just in front of the precentral convolution and immediately beneath the cortex.

Just as this paper was approaching completion an article by V. Plavec on minor (smaller) motor epilepsy appeared in the *Neurol. Centralbl.*, Nos. 3, 4 and 5, Feb. 1, Feb. 15 and March 1, 1906. The author contributes the history of a case of a boy eleven years old at the time of the first observation. At this time the diagnosis of facial tic was made. Some years later the patient again fell under the attention of Plavec, when the diagnosis of abortive epilepsy was made, and still later it became clear that the case was one of ordinary idiopathic epilepsy with the minor or local seizures being similar in character to the Jacksonian attacks which are observed to occur during the progress of a true idiopathic convulsion. The not very extensive continental literature of the subject of Jacksonian epilepsy is reviewed by Plavec, especial attention being given to articles by Féré,[8] Binswanger,[9] Burnhardt,[10] Sarrailhe,[11] Liebert[12] and Kjelmann.[13]

He defines smaller motor epilepsy as that form of attack in which a local spasm occurs without disturbance of consciousness and without an aura. The differential diagnosis of hysterical attacks, reflex spasms, seizures due to cortical

[8] Féré: Revue Neurolog., 1901, S. 143; also Épilepsies et épileptiques, Paris, 1890, Alcan., S. 104.

[9] Binswanger: Die Epilepsie in Nothnagel's Pathologie u. Therapie, xii, S. 148

[10] Burnhardt: Erkrankungen der peripheran Nerven. *Ibid.*, xi, S. 41.

[11] Sarrailhe: Revue neurolog., 1903, S. 668.

[12] Liebert: Deutsche med. Wochenschr., 1885, No. 37.

[13] Kjelmann: Berliner klin. Wochenschr., 1894, No. 13.

lesions and idiopathic epilepsy is considered at some length in the light especially of the valuable contributions of Binswanger and Féré. In connection with what has been said in this paper, the conclusions of Féré, that cases of so-called tic are often in reality of epileptic nature, are of interest. Cases are cited to demonstrate the truth of this position. So-called attacks of myoclonus probably occur in about 5% of the cases of idiopathic epilepsy. The reader is referred to the paper of Plavec and also to the contributions of Binswanger, Féré and others for some interesting points in the diagnosis of Jacksonian epilepsy and its counterfeits.

A few of the differential points brought out by these writers might be presented here in addition to those to which attention has been called in previous pages.

The aura in idiopathic epilepsy is not as common as that occurring in organic epilepsy and in those cases of epilepsy due to reflex causes. Idiopathic epilepsy is liable to occur at night; true tics and hysterical attacks scarcely ever at night, although one or two cases are noted in the literature in which severe cases of tic were said to have occurred at night. Reflex epilepsy and true Jacksonian attacks may occur at night. Féré and Binswanger believe that idiopathic epilepsy may be of organic origin and that Jacksonian attacks may be only part of the idiopathic attacks, a point which I have already somewhat fully considered. Jacksonian attacks do not come on as often at night as do the idiopathic attacks.

Féré states that in Jacksonian or cortical epilepsy the patient is always awakened from sleep so that he is compelled to lie awake during the whole attack; in idiopathic epilepsy the patient is only awakened from sleep in those attacks which, during the day, would not cause him to lose consciousness.

An important symptom of minor motor epilepsy is more or less paralysis of the part after an attack. This occurs most often after cortical or organic epilepsy, but cases are on record in which after a reflex or idiopathic attack this has been present. The paresis may be overlooked, as it may last only a few minutes after the attack. At times instead of the convulsion the epileptic attack may be manifested by weakness, temporary in character, and this may occur without loss of consciousness. Instead of paresis there may be sensory changes, as diminution of the visual fields and disturbances of cutaneous sensation.

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THE
FOCAL DIAGNOSIS OF OPERABLE TUMORS OF THE CEREBRUM.[1]

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INTRODUCTION.

IN 1888, before the Congress of American Physicians and Surgeons, I presented a paper on cerebral localization in its practical relations, in which the status of focal brain surgery was discussed, the article including a consideration of the removal of brain tumors, and also of cortical excision and the operative treatment of hemorrhage, abscess and other lesions amenable to surgical procedure.[2] About this time and during a few succeeding years, the application of the principles of cerebral physiology to practical surgery attracted the attention and excited the enthusiasm of the profession in Europe and this country. Enthusiasm was dulled and attention relaxed by frequent failures in the attempts to exactly localize lesions, and by the difficulties which beset the surgeon in his efforts to remove these lesions with the trephine and rongeur.

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[1] Read before the meeting of the Medical Society of the State of Pennsylvania, held at Scranton, Pa., September 26, 27 and 28, 1905.

[2] Transactions of the Congress of American Physicians and Surgeons, vol. i., 1888; also Brain, vol. xii., 1889.

Since the introduction of the osteoplastic operation and the newer researches into cerebral localization which have more clearly defined the motor and sensory regions of the cortex and the subdivisions of the association areas, the surgery of the brain has received a new impetus, the effects of which are seen in the increasing number of successful cases of operative interference reported in the last five or six years.

In a series of papers on localization and the neurology and surgery of brain tumors published during the last five or six years, I have given the results of many personal investigations.[1] This article is intended to summarize the

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[1] Tumor of the Superior Parietal Convolution, Accurately Localized and Removed by Operation. Journ. of Nerv. and Ment. Disease, May, 1900. (With Dr. W. W. Keen and Dr. Wm. G. Spiller.)

The Localization of Brain Tumors, Especially with Reference to the Parietal and Prefrontal Regions. Phila. Med. Journ., April 20, 1901.

The Separate Localization in the Cortex and Subcortex of the Cerebrum of the Representation of Movements and of Muscular and Cutaneous Sensibility. Journ. of Nerv. and Ment. Disease, November, 1901.

Tumor of the Brain Localized Clinically and by the Roentgen Rays, with Some Observations and Investigations Relating to the Use of the Roentgen Rays in the Diagnosis of Lesions of the Brain. Phila. Med. Journ., February 8, 1902. (With Dr. G. E. Pfahler.)

An Additional Case of Tumor of the Brain Localized Clinically and by the Roentgen Rays. Phila. Med. Journ., September 27, 1902. (With Dr. G. E. Pfahler, and Dr. J. B. Deaver.)

A New Scheme of the Zones and Centres of the Human Cerebrum, with Remarks on Cerebral Localization, Especially with Reference to Osteoplastic Operations for Brain Tumor. Journ. of the Amer. Med. Association, October 4, 1902.

The Surgery of Brain Tumors from the Point of View of the Neurologist, with Notes of a Recent Case. Phila. Med. Journ., Nov. 29, 1902.

The Physiological Areas and Centres of the Cerebral Cortex of Man, with New Diagrammatic Schemes, UNIVERSITY OF PENNSYLVANIA MEDICAL BULLETIN, May, 1904.

A Glance at the History of Cerebral Localization, with Some Considerations Regarding the Subdivisions of the Areas of Representation of Cutaneous and Muscular Sensibility and of Concrete Concepts. Proceedings of the Phila. Co. Med. Soc., September 30, 1904.

Subdivisions of the Concrete Concept Area of the Human Cerebrum. The Med. News, November 5, 1904.

The Diagnosis of Tumors of the Cerebellum and the Cerebello-Pontile more detailed studies of the papers referred to, presenting at the same time some new views and observations. It has been found necessary in several instances to change the positions and extensions of centres and areas as formerly given in accordance with newly obtained data. This is more particularly true with regard to the position and subdivisions of the motor area, a subject which has received full consideration at the hands of Dr. Frazier and the writer in a recent paper in the UNIVERSITY OF PENNSYLVANIA MEDICAL BULLETIN.

GENERAL SYMPTOMS.

The general symptoms of brain tumor, which for completeness should receive brief preliminary consideration, are, as is well known, headache, vertigo, nausea or vomiting and optic neuritis. All of these may be present at one time or at some period during the progress of the case. In rare instances all of them may be absent, or one, two or any number of them may be present or absent. Their absence, severally or together, is therefore not conclusive against the existence of an intracranial growth.

The localization of headache cannot be depended upon as an indication of the site of a tumor. While the pain in the head caused by the presence of a brain tumor is often severe and may be agonizing, the complete or almost complete absence of headache is at times remarkable. This absence of headache is not always to be explained by the tumor being soft or deep-seated. In a recently observed

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Angle, Especially with Reference to their Surgical Removal. New York Med. Journ. and the Philadelphia Med. Journ., February 11 and February 18, 1905; also Philadelphia Hospital Reports, vol. vi., 1905.

The Motor Area of the Human Cerebrum; its Position and Subdivisions, with Some Discussion of the Surgery of the Motor Region. UNIVERSITY OF PENNSYLVANIA MEDICAL BULLETIN, July–August, 1905. (With Dr. Chas. H. Frazier.)

case of tumor of large size and moderate firmness, the growth being adherent to the membranes, pain in the head was never suffered. Most frequently headache is absent in those cases of tumor which are subcortical and infiltrating, and of a consistency not much different from that of the brain itself. It follows that while the presence of severe pain in the head is strongly confirmative of the diagnosis of brain tumor, its absence cannot be regarded as excluding this diagnosis.

The same statement might be made with regard to optic neuritis, although it is more frequently absent than headache, at least this has been my own experience. It is probable that optic neuritis would almost always be found if the patient remained under observation until death, especially if operation was not performed. The reader is referred to Dr. G. E. de Schweinitz's paper on the "Ocular Symptoms of Tumor of the Cerebrum," published in the UNIVERSITY OF PENNSYLVANIA MEDICAL BULLETIN for April–May, 1906, for a full discussion of this subject. Vertigo is rarely absent, but is often so little expressed or so little taken account of by the patient as to not attract the attention which it deserves. Nausea and vomiting, although often present and frequently presenting typical features, as for instance, their occurrence without accompanying signs of gastrointestinal irritation, are, on the whole, at least for long periods, as often absent as present.

When headache, optic neuritis, vertigo and nausea and vomiting are all present and persistent in the same case, the tumor is likely to be connected with the dura, and therefore accessible if situated anywhere on the brain convexity, the exceptions being cases of subcortical tumor of large size and great density.

A word might be said about the comparative importance of general and focal symptoms. The latter may, on the whole, be regarded as the more important of the two, although the presence of a general symptom, such as optic neuritis, may be the firmest link in the chain of evidence. Vertigo and nausea and vomiting are never absolutely conclusive, although they are not infrequently the symptoms which first suggest the existence of a tumor.

BRAIN TUMORS, PHYSIOLOGICAL AREAS AND SURGICAL OPENINGS.

Tumors and vascular lesions, such as hemorrhage, thrombosis, and embolism, tend to focus in special physiological areas. A tumor nearly always starts in what might be termed the heart of such an area, and largely occupies one such physiological region to the exclusion of others, although of course it may invade in any direction from its initial focus.

Following the old anatomical subdivisions of the brain into frontal, parietal, occipital and temporal lobes, these lobes can be conveniently subdivided for the purpose of diagnostic study and operative procedure into six areas, as follows: (1) the prefrontal or higher psychic; (2) the midfrontal or intermediate psychic-motor; (3) the postfrontal or motor; (4) the parietal or sensori-stereognostic; (5) the parieto-temporal or visuo-auditory; and (6) the occipital or visual. Each of these operable regions has its special symptom complex. It has also, from the surgical side, the special opening which will uncover it to best advantage.

With regard to the designation intermediate psychic-motor which has been applied by me to the second or midfrontal area, while this term is not altogether unobjectionable, it serves to express the idea that the functions of this region are intermediate between those of the purely motor zone and the region of higher psychic activity. Skilled movements of the upper and the lower extremity and of the musculature concerned with speech which are represented in this midfrontal zone, require constant guidance of purely motor stimuli by higher mental faculties as attention, comparison and reasoning, which are represented in the prefrontal lobe.

THE DIFFERENCE BETWEEN THE FUNCTIONS OF THE RIGHT AND LEFT CEREBRAL HEMISPHERES.

In the consideration of focal cerebral diagnosis some attention must be paid to the subject of the difference between the left and the right hemispheres. In the human race the left hemicerebrum, with rare exceptions, is the leading half of the brain, and as a rule the higher the individual development, the greater will be the disparity between the two hemispheres in favor of the left. In children this difference is not so marked as in those of more mature years, and in the laboring classes not so decided as in those who pursue mechanical arts requiring special skill, while in the latter it is not so pronounced as in those of high intellectual capacity and acquirements. With regard to the last two classes, however, it must be remarked that in those who pursue mechanical arts or other vocations chiefly concerned with the concrete, the concrete memory field of the cerebrum may proportionately have a higher development than in those whose pursuits are esthetic, philosophical or ethical. In the latter the prefrontal or higher psychic region has its highest evolution.

It is scarcely necessary to do more than allude to the preponderant part played by the left hemisphere in the faculty of speech, and to call attention to the corollary that while lesions of this hemisphere, according to their location, may destroy respectively or conjointly the forms of sensory, conceptual or motor speech, those of the right hemisphere may leave these functions untouched or only transiently affected. The exceptions in the left-handed, or even in extremely rare cases in others, must of course be borne in mind.

In the case of speech and writing, the conclusions in the pages which follow are largely for the left hemisphere alone. Motor aphasia, motor agraphia, word blindness, word deafness, paraphasia, paragraphia, dyslexia, alexia, anomia and other disorders of speech and writing constituting parts of the focal syndromes which will be presently considered are to be referred entirely to disease of the left hemisphere. It is for this reason that tumors and other lesions of the right hemisphere are sometimes not so easy of focal diagnosis as those of the left. While destructive lesions of the left temporal lobe give symptoms readily traced to their anatomical seat, the right temporal lobe is one of the most latent regions of the brain.

The remarks here made, with regard to the difference between the functioning of the left and the right hemispheres, have their least application in a study of the local symptoms resulting from tumors situated in the primordial zones. Symptoms referable to the motor zone as now defined by the writer, namely, that region chiefly represented by the precentral gyre and the precentral portion of the paracentral lobule, have for the purposes of focal diagnosis almost equal value for either the right or the left side; in other words, a lesion of either the right or the left precentral gyre will give contralateral symptoms of definite type. In like manner a lesion of the primary or lower visual area, that is, of the calcarine fissure and cuneus of either side, will give almost identical contralateral symptoms. The same is true of lesions of the primary auditory olfactory and gustatory centres, remembering with regard to the primary auditory centre that word hearing is not a function of this centre, but of the higher auditory area contiguous to it, this being a part of the concrete concept area. The cortical representation of cutaneous sensibility and the muscular sense may be somewhat more highly evolved on the left than on the right side, but in the main it is true of the areas supplied with projection fibres that their cortical centres are equally developed in each hemisphere. Some facts seem to indicate that in the case of common sensibility one hemisphere has a larger power of representing both sides of the body than in the case of other functions.

Object recognition has a higher evolution in the left hemisphere than in the right, although both hemispheres take large part in this function. It follows that destruction of both occipital lobes or of parts connecting them is necessary for the production of complete and permanent mind blindness, and yet transient mind blindness may occur from a large lesion in the left occipital region.

Both prefrontal regions take part in higher psychic activities, but carefully collected data regarding lesions of the prefrontal lobes indicate that the left prefrontal region plays a more important role in intellection than the right.

I have closely studied the symptomatology of special cases of lesion of the left and the right parietal lobes, and in a previous paper have stated my belief that stereognostic conception probably has a higher evolution in the left hemisphere. A lesion of the posterior and superior portion of the right parietal lobe may therefore not give astereognosis of so decided a character as one in a corresponding position in the left parietal lobe, although the stereognostic function must have considerable development in both hemispheres.

From what has just been said about the difference between the functional power of the two hemicerebrums, this must be taken into most account when considering tumors or other lesions presumably present in the lower, intermediate and higher association areas of the cerebrum.

GENERAL REMARKS ON THE DIFFERENTIATION OF CORTICAL AND SUBCORTICAL TUMORS.

A brain tumor, like most massive lesions of the brain, is rarely strictly limited either to cortex or subcortex. If the neoplasm begins in the subcortex as a rule it spreads eventually to the surface; if, on the other hand, it originates in the dura or in the pia arachnoid it first compresses and then destroys the cortex and sooner or later destructively invades the subcortex. The clinical consequence of this joint involvement is that usually a sharp line of demarcation cannot be made between the symptoms of cortical and subcortical tumors at the time when the cases are under observation; nevertheless, a tumor springing from the membranes gives early and predominating cortical symptoms, while one arising in the centrum ovale gives early and predominating subcortical symptoms. The localizing symptoms of each subcortical region are correlated with those of the physiological cortical area to or from which the fibres of the subcortex come or go.

The question of the diagnosis of subcortical growths is one of great importance. Of course, speaking generally the term subcortical is applicable to all growths situated below the cortical cinerea, and in this sense might include not only tumors of the corona radiata and internal capsule, but also those of the basal ganglia, crura, pons and oblongata. These subcortical regions must, however, be separated, in order to come to any practical conclusions in differential diagnosis.

It is necessary therefore to distinguish two classes of coronal tumors—those which grow from within outward and those which invade from the cortex inward. The latter include entirely accessible tumors and will later be fully considered. As the subcortex is destructively invaded, the symptoms at first transient and recurring, such as agraphia, aphasia, paresis, astereognosis, word blindness or word deafness, become more and more persistent and permanent. As each separate cerebral area is discussed, some attention will be paid to the differentiation of tumors predominatingly cortical or subcortical.

It may be said that tumors, like other lesions of the corona radiata, will present variations in their symptomatology according to the relative number and particular value of projection, association, and commissural fibres in the part of the subcortex which is the seat of disease—a point to which some consideration will necessarily be given in the remarks on differentiation. Where projection fibres predominate, as in the subcortex of the motor and of the visual auditory and other regions of the special senses, the symptoms referable to irritation or destruction of these fibres will overshadow in importance in the study of the case those referable to implication of the fibres of association, and the focal diagnosis will chiefly depend upon the interpretation of the former. In the great association areas, posterior and anterior, the sensorial and motor symptoms will be more or less relegated to the background, although in the case of a tumor, this not infrequently involves some part of the projection system, as for instance the optic radiations, because of the manner in which they pass from the basal centres to their primary cortical destinations. Peculiar variations in symptomatology, no doubt, arise because of the disruption of the commissural connections between the two hemispheres. through the callosum and commissures; but the symptoms of such lesions rarely play an important part in practical focal diagnosis, although some exceptions to this statement need to be noted, as when considering such symptoms as cortical and subcortical word blindness, or such a question as that of object blindness being due to a lesion of one or both hemispheres or of the tracts uniting these hemispheres.

TUMORS OF THE PREFRONTAL LOBE, THE HIGHER PSYCHIC REGION.

Beginning at the anterior pole of the cerebrum, the first physiological region of diagnostic and surgical importance is one concerned with the highest functions of the brain. On the lateral aspect of the hemicerebrum it reaches from the anterior tip to a line drawn from a point about half an inch in front of the anterior branch of the Sylvian fissure (the pre-Sylvian fissure) nearly vertically to the mesal edge of the hemisphere. Its posterior boundary is therefore the midfrontal or intermediate psychic motor region, occupying the posterior portions of the first, second and third frontal gyres. Tumors if destructive, or other destructive lesions of the prefrontal lobe, and especially of the left prefrontal, will cause symptoms indicative of interference with the highest psychic functions. One of the chief difficulties in determining the exact functions of this region has resulted from inaccurate or imperfect observation of mental symptoms due to lesions situated in various portions of the brain.

It has occurred to me several times to examine patients suffering from brain tumor, in whose cases psychic symptoms have been noted, and in a few instances have been given localizing value, but without due consideration. The pain, distress, irritability, inattention, and mental confusion so commonly caused by a brain tumor wherever situated are set down as symptoms due to disturbance of the higher psychic functions, while in fact they are simply the expression of exhaustion and physical suffering, or of these together with impairment of concrete memories and powers of expression. The symptoms which are really referable to destructive lesions of the prefrontal portions of the cerebrum are best expressed by stating them to be evidences of higher psychic dissolution, which is shown in loss of memory and of powers of attention, abstract conception, comparison, judgment and higher imagination.

As in this contribution I am concerned almost exclusively with the focal diagnosis of brain tumor with the view of fixing the site of operation, I shall not go at any length into a discussion of disputed points in cerebral localization. Recently, in a paper with Dr. T. H. Weisenburg,[1] the question of the localization of the higher psychic functions with especial reference to the prefrontal lobe is discussed with the report of a case of brain tumor with necropsy, the record of which favors the view of the existence of a higher prefrontal psychic area. This subject has also been discussed by me in other publications.

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[1] Chas. K. Mills and T. H. Weisenburg, Journal of the American Medical Association, vol. xlvi., Feb. 3, 1906.

The morphological and anatomical observations show that the cephalic extremity of the cerebrum in the human being is concerned with functions of higher psychic valuation than that of any other cerebral region. Embryological investigations show the absence of projection cells and fibres in a region structurally rich in cellular association systems of an unusual order.

Many clinico-pathological investigations apparently contradictory to the view here expressed regarding the functions of the prefrontal lobe have been adduced, from the famous American crow-bar case to the most recent so-called digests of cases of brain lesion showing psychic symptoms. These can be passed by for the present with the assertion that they are not conclusive, which appears the more evident the more closely the compilations are examined. Cases confirmative of the view that the prefrontal region and especially the left prefrontal is concerned with the highest psychic functions have been collected in large number.

In order that any decided effect upon the mentality of the individual shall result, the lesion of the prefrontal lobe must be destructive and must be of some extent. A tumor growing from the dura may produce no psychic symptoms of importance except those which are the result of irritation or tension until it has destructively invaded the brain substance.

Such destructive invasion occurs as soon as occluded or obliterated vessels cause necrosis of brain tissue. The victim of such a destructive prefrontal lesion begins to show failure of memory which becomes more and more decided as the disease advances. This failure of memory relates more to matters abstract, general and distant than to those connected with his immediate surroundings. The power of attention, and especially of sustained attention, may be decidedly impaired from an early period. Problems formerly easy to solve become impossible. Inhibition is impaired. Hesitation, uncertainty, lack of force, slowness in apprehending and in communicating ideas, and motor disquietude due to loss of control, may be present.

Tumors of the orbital surface of the frontal lobe give especially psychic, olfactory and optic tract symptoms.

Some attention should be paid in this connection to the not infrequent occurrence of indirect and distant symptoms of a pronounced character. A frontal or prefrontal tumor may give severe occipital pain or the reverse may happen —a cerebellar growth causing frontal pain. Cerebellar or cerebellopontile symptoms have been noted in a few cases of prefrontal tumor, these being of large size and of such density as to exert considerable pressure. Collier,[1] for instance, has called attention to such a case of prefrontal tumor in which the only bulbar lesion present was degeneration of the eighth nerve. The patient's main symptoms after the disease had existed for seventeen months were paralysis of the left external rectus, marked nystagmus with a slow movement to the left, complete left nerve deafness, left peripheral facial paralysis, left cerebellar position of the head (head inclined to left shoulder and face rotated to the right), marked head retraction during paroxysms of pain, bilateral ataxia with lurching to the left, and diminished right knee-jerk. Necropsy revealed glioma of the right prefrontal lobe. The cerebellum especially the left lateral lobe was much indented into the edge of the foramen magnum.

While attention is called to the extraordinary symptomatology of this case, and while the possibility of the occurrence of a similar case with a like symptomatology should be borne in mind, too much stress should not be laid upon it in the study of focal diagnosis.

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[1] **James Collier, The False Localizing Signs of Intracranial Tumor, Brain, 1904, part iv.**

I have never seen a well-marked case of a so-called frontal ataxia, although it doubtless exists and may be due, as has been suggested, to involvement of the fronto-cerebellar tracts. Cases of agenesis and of lesions of the prefrontal lobe with some ataxia have been recorded. When present, it may be a symptom of inattention or of lack of judgment with regard to the movements rather than a true ataxia such as is seen in cerebellar or in postparietal disease. At a late period in the history of the case of prefrontal tumor which I reported with Dr. Weisenburg, the patient had a shuffling gait and was unable to convey food to his mouth with any certainty. Soon after he began to have muscular tremors in his hands and legs much like those seen in paralysis agitans.

On several occasions at periods separated by months, I saw in consultation a case which seemed to have some points in common with the one recorded by Collier, and which was eventually proved by operation and necropsy to be one of tumor of the right prefrontal and midfrontal region; it was situated so that its posterior limits grazed or merged into the motor region. The cerebellar and cranial nerve symptoms may have been due in part to counterpressure and traction.

The most important symptoms in this case were: (1) general enfeeblement; (2) what appeared to be bulbar and other cranial nerve symptoms, including dilated left pupil, sluggish iritic reflexes, left ptosis, and other third nerve palsies, nasal voice, some difficulty in swallowing, facial paresis on the left, drooling, and partial atrophy of the tongue; (3) ataxia with paresis of left upper extremity, paresis of right lower extremity, probable ataxia of left lower extremity, and inability to walk or even stand without support; (4) marked Babinski reflex on the right and normal, although not overprompt, plantar reflex on the left; and (5) impairment or loss of control over bladder and bowels. This patient on several occasions at long intervals had attacks of Jacksonian epilepsy, the spasm being usually most marked in the left side of the face and in the left upper extremity.

With regard to the prefrontal lobe, as this terminal zone contains no projection fibres, the symptomatology of tumors of the cortex and subcortex will not differ sufficiently to allow marked clinical distinctions to be made. Tumors of the membranes, as already indicated, simply compress the prefrontal lobe and may not exhibit a symptomatology of sufficient distinctness to allow them to be diagnosticated. The more and more the prefrontal subcortex is destructively invaded the more and more serious will be the symptoms of higher psychic dissolution.

TUMORS OF THE MIDFRONTAL OR INTERMEDIATE PSYCHIC-MOTOR REGION.

A tumor occasionally develops with its initial nidus in some part of what has been designated in our scheme as the midfrontal or intermediate psychic-motor region. The physiological centres and areas included in this region are the speech centre of the hinder part of the third frontal convolution, the graphic centre in the posterior part of the second frontal convolution, and the centres for movements of the head and eyes, intermediate between the speech and writing centres. A case of tumor involving this region was recorded by McConnell.[1] The symptom complex in such a case, if the left hemicerebrum be involved, usually has for its central phenomena motor aphasia, motor agraphia, and Jacksonian epilepsy, with movements of the head or of the head and eyes as a salient feature of the local spasm. The Jacksonian epilepsy includes spasm of the facial musculature and movements of the upper extremity. Some psychic symptoms of a special sort may be present, these being attributable to involvement of the prefrontal region. In McConnell's case, the patient's power of sustained attention and his memory were transiently affected. If the motor region is not directly involved, paralysis of the face and of the extremities is not present, or only to a slight extent. In McConnell's case some facial paralysis was detectable, but the arm, although decidedly affected in the spasmodic attacks, showed scarcely any impairment of motility. In this case the growth was successfully removed by Dr. Chas. H. Frazier, the patient recovering from his agraphia and his moderate facial articulatory paresis. His mental symptoms also cleared up entirely.

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[1] J. W. McConnell, UNIVERSITY OF PENNSYLVANIA MEDICAL BULLETIN, July-August, 1905.

The difference of opinion and the disputes which have arisen regarding the existence of a separate motor graphic centre probably have their origin in part in misapprehensions of exactly what is meant by such a centre. Agraphia like aphasia may arise from lesions situated in different functional regions of the cerebrum. A cortical visual agraphia may, for instance, be due to a lesion of the centres for word seeing and letter seeing in the angulo-occipital cortex. An imperfect agraphia or paragraphia may be dependent upon destruction of tracts connecting the visual with the auditory centres, or connecting both of these centres with Broca's convolution. A form of agraphia results from a lesion of Broca's convolution. An aphasic who cannot arrange language for expression cannot write what he is unable to say, or can only do this very imperfectly. He may, however, be able to copy if sufficient time and care are taken. In one of my cases, the patient who had, as shown by necropsy, a lesion of the hinder part of the third frontal convolution and of the insula, could only write to a limited extent, but what he wrote was correctly spelled and expressed. If the posterior portion of the second frontal convolution is destroyed, the form of agraphia which results differs from that which is observed in the other instances just recited. The patient who may retain full powers of movement in his arm, forearm, hand and fingers, supposing his precentral convolution to be undestroyed, is not able to use these parts skilfully for the purpose of writing, and usually for other graphic purposes, as for drawing. The cerebral centre which controls the arrangement and coördination of these movements no longer exists, although the centre for the coarse movements, out of which they have been developed, may still be intact, as may also the centres for the arrangement and coördination of articulate speech.

In a case of midfrontal and postfrontal tumor seen by me in consultation with Drs. Weir Mitchell and John K. Mitchell, and operated on by Dr. Keen, agraphia was one of the most interesting symptoms. Other symptoms present were attacks of temporary aphasia, some evidences of psychic loss or change, twitchings, first of the right and then of the left side of the face, a few convulsive attacks and slight enfeeblement of the right arm. All the forms of visual and auditory aphasia, hemianopsia and disorders of sensibility were absent. Operation revealed a tumor which was of large size. The patient died of shock and hemorrhage, and a subsequent careful examination was made of the position and extensions of the tumor by Dr. William G. Spiller. The tumor was an endothelioma measuring 7 by 8 cm. The upper part of the precentral and the highest part of the first frontal convolutions were not compressed by the tumor. The lower posterior portion of the first frontal and the whole of the posterior portions of the second and third frontal convolutions and the lower two-thirds of the precentral except the extreme lowest part were all compressed beneath the growth. The tumor did not extend behind the central fissure and did not invade the brain tissue, but started apparently from the dura, pressing upon and causing thinning of the cortex and localized atrophy of the cerebrum.

In this case the agraphia present was peculiar and was briefly described by me in a letter to the doctors Mitchell, as follows: "The agraphia is certainly the most interesting clinical feature. In general terms it belongs under the head of motor agraphia, and a study of his writing gives some support to the view of those who have said that the so-called motor graphic centre might, perhaps, be better called an orthographic centre. He tends to repeat letters, syllables and words in writing, misspelling both common and proper names, evidently at times recognizing his errors and attempting by erasures and rewriting to correct them."

This case has been reported by Gordinier[1] from notes furnished by Dr. John K. Mitchell.

Tumors strictly limited to the hinder portion of the left third frontal convolution are rare, but such cases have been recorded. A tumor may originate in the preinsular portion of the Sylvian fossa, involving in its progress the third frontal and, perhaps, other contiguous regions. Such a case, while comparatively easy of diagnosis and accessible, may be difficult or impossible to remove. The effort should, however, be made and may be rewarded with success if the neoplasm should prove to be an enucleable dural growth. If the primary effects of the growth should be in the third frontal gyre the chances of success would be greater.

While tumors strictly limited to Broca's convolution are rare, this cerebral region is frequently involved by invasion. A growth beginning in the motor region may advance cephalad so as to involve both the second and third frontal gyres, or a prefrontal tumor may gradually encroach upon the posterior portions of the third, second and first frontal convolutions or either of these. The initial and the invasion symptoms must always be carefully studied in such cases.

Presuming the rare case of a tumor confined almost entirely to the third frontal gyre and preinsula, the symptoms will be those long described under the name of motor aphasia. The patient according to the destructive extent of the lesion will be unable to recall and arrange for use words, phrases and sentences. He may retain a few words or expressions, or have only a single recurring utterance; he may be able to write in proportion to the degree of his ability to arrange language for emission or projection. If the growth is strictly limited to the third frontal and insula he will not have agraphia of the form which is observed from destructive lesion of the graphic centre in the posterior portion of the second frontal. Even when the growth is somewhat closely confined to Broca's convolution some pressure symptoms may be present, and these will usually be in the form of paresis of the face or upper extremity. Jacksonian epilepsy of the aphasic-motor type may appear, and in this case the spasm will usually be ushered in by some repetitive speech disorder followed by facial or faciobrachial spasm, or even by hemispasm.

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[1] H. C. Gordinier, American Journal of the Medical Sciences, September, 1903.

A few words might be said here about the effect of the involvement in a tumor or other lesion of the centres for movements of the eyes or of the head and eyes which lie between the graphic and motor speech areas, and to some extent in advance of them. Destruction of these centres interferes with the performance of skilled movements of all sorts. The part usually played by movements of the eyes and head in reading, speaking, writing, drawing, engraving, painting and similar pursuits will be disturbed. The guidance of skilled movements of the lower extremity such as are used in dancing, and of both the lower and upper extremities, as for instance those employed in fencing, will be impaired or lost.

The midfrontal region containing the centres for skilled and highly evolved movements of the upper extremity, of the head and eyes, and movements concerned with speech, probably is not furnished with projection fibres, although this question is not entirely settled. The centres for writing and speech probably have short neuronal associations on the one hand with the precentral motor centres, and on the other with the higher psychic centres of the prefrontal lobe; also of course they are associated with the sensory and concept centres of the cerebral zone of speech. The connections of these regions with the articulatory, phonatory and motor centres at the base of the brain and in the spinal cord are probably indirect through the centres of the motor zone.

The symptoms produced by midfrontal subcortical tumors can, it will be seen, scarcely be differentiated from cortical growths. Both will give clinical phenomena largely dependent upon the disruption of associations with other regions—psychic, motor and sensory. Aphasia, agraphia and disorders of the movements of the eyes and head will be present both in cortical and subcortical growths if these are of considerable dimensions. A tumor of the membranes invading the midfrontal cortex will however exhibit agraphic and aphasic symptoms of irregular and oscillating development, the patient retaining his powers of speech and writing later than in a case of destructive midfrontal subcortica tumor.

TUMORS OF THE POSTFRONTAL OR MOTOR REGION.

In the article by Dr. Frazier and the writer, already referred to, giving the results of faradization of the human cerebrum, the position is taken that the motor area is situated entirely cephalad of the central fissure, and is largely confined to the precentral convolution, its only extension beyond being an area for movements of the eyes and head in contiguous portions of the posterior extremities of the second and third frontal gyres. A large percentage of cerebral tumors originate in this region. It is scarcely necessary to discuss in much detail their focal diagnosis. The main symptoms are paresis, deepening into paralysis of the face, arm and leg, or of two or more of these portions of the body. The upper extremity is most frequently and most profoundly affected, movements of the leg, face, jaw and of the head and eyes being impaired in extent and degree in the order of the parts named.

At an early stage of the disease monospasm usually appears, and in most cases this has a distinct initial or signal symptom. A spasm may at first be confined to the part in which it begins, as to one side of the face, to the thumb, to the thumb and fingers, or to the thumb, fingers and wrist. Occasionally the initial symptom is in the lower extremity, showing itself, for instance, in a movement of the great toe or in an extensor or flexor movement of the foot. Almost invariably, as the tumor grows and the case develops, the spasm spreads. Facial spasm may for example have added to it spasm of all parts of the upper extremity; or convulsive movements beginning in this extremity may extend so as to involve either the leg, the face, or the eyes and head, or even all of these parts. The convulsion in the further progress of the case may become general, radiating to the other side.

Agraphia and aphasia on the one hand, or cutaneous and muscular paræsthesia and anæsthesia on the other, may be invasion symptoms.

Tumors strictly confined to the centrum ovale of the motor region are rare. A tumor beginning in the motor subcortex and up to the time of observation limited to it, will in the first place give symptoms of paresis or paralysis early, and these will be associated with tonic spasticity, Jacksonian spasms usually not being present. The tendency seems to be for spasms of the Jacksonian type to show themselves as soon as the motor cortex is invaded, either from within or from without, but this rule is not absolute, and a subcortical motor growth may give paroxysmal monospasm or hemispasm. When the tumor simply irritates the cortex, Jacksonian spasm may be present, although well-defined paralysis and tonic spasticity are absent; when the lesion is largely subcortical both paralysis and spasticity are marked clinical phenomena. A motor subcortical tumor is more likely than a cortical growth to have sensory symptoms associated with the disorders of motility, because the further the growth is removed from the surface of the brain the closer becomes the relations of motor and sensory tracts entering and emerging from the internal capsule. Reflexes in subcortical motor growths, as in cortical lesions, are exaggerated, but the ankle clonus and the Babinski response are if anything more persisting and striking phenomena in subcortical than in cortical lesions.

A tumor strictly confined to the motor region does not give objective sensory phenomena of a persisting character; the localizing symptoms of a growth so situated are motor, chiefly, as just described—paresis, paralysis and monospasm. Both the superficial and the deep reflexes on the side opposite to the lesion are abnormally exaggerated, much increased knee jerk, persistent ankle clonus and the Babinski reflex being commonly present. Hemianopsia, object blindness, speech disorders and true psychic symptoms are absent unless the tumor has greatly extended beyond the motor region.

In fixing the site of a tumor of the motor zone Jacksonian epilepsy plays an important diagnostic role, scarcely second in importance to monoplegia or hemiplegia. The significance of this form of spasm in focal diagnosis and the site and nature of the lesions causing it have been made by me the subject of another paper, to which the reader is referred.[1]

I shall summarize here a few of the most important points of this contribution. The term Jacksonian epilepsy is used in a broad sense as indicating monospasm or hemispasm due to cortical or subcortical discharge. It is by no means always due to gross lesion of the motor zone, although often an important sign of such lesion.

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[1] Chas. K. Mills, The Significance of Jacksonian Epilepsy in Focal Diagnosis, with Some Discussion of the Site and Nature of the Lesions Causing this Form of Spasm. The Boston Medical and Surgical Journal, April 26, 1906.

In hemiplegias due either to softening or hemorrhage, the cortex surrounding the gross lesion is often the seat of congestion and small extravasations, which irritating the brain substance and rendering it unstable, cause epileptiform attacks. These may take the form either of monospasm or of hemiepilepsy, and may be improperly regarded as caused by a tumor.

Many cases have been recorded in which Jacksonian epilepsy has been used in fixing the site of a tumor. Some of these cases reported by others or myself have been operated on successfully by Dr. Chas. H. Fraizer, Dr. W. W. Keen, Dr. W. J. Hearn, and other Philadelphia surgeons. In all of these cases, in addition to the Jacksonian spasm, other symptoms of tumor of the motor zone, such as monoplegia, exaggerated deep reflexes and the Babinski response were present; and if the cases were strictly localized to the motor area, sensory and psychic phenomena, and speech, visual and auditory disturbances were absent. The general symptoms of brain tumor have usually been present, although I have seen cases of tumor of the motor zone in which headache and optic neuritis were not among the symptoms.

With regard to the seat and nature of the lesions and disturbances causing Jacksonian epilepsy, it must be borne in mind (1) that tumors situated in other parts of the brain than the motor cortex may cause Jacksonian epilepsy; (2) that other lesions beside tumors situated in the motor cortex may cause this form of spasm; (3) that it may occur in toxic and other diseases in which no demonstrable focal lesions are present; (4) that a spasm closely counterparting the Jacksonian type may be observed as a reflex or an hysterical disorder; and (5) that Jacksonian epilepsy may be simply an integral part or the entire expression of a case of idiopathic epilepsy. Each of these is fully considered in the paper to which reference has just been made.

TUMORS OF THE PARIETAL LOBE, REGION OF REPRESENTATION OF CUTANEOUS AND MUSCULAR SENSIBILITY AND OF STEREOGNOSTIC CONCEPTION.

In this connection the parietal lobe is defined after the usual manner of the anatomical works, namely, considered as regards the lateral aspect of the hemisphere, as that part of the cerebrum which is situated between the central fissure in front, the line of the parieto-occipital fissure behind and that of the Sylvian fissure below. It includes, therefore, the postcentral, superior parietal and inferior parietal gyres. This lobe can be divided physiologically into areas of representation of cutaneous and of muscular sensibility and of stereognostic conception, the first having its seat in the postcentral gyre; the second probably in the anterior portions of the superior and inferior parietal convolutions, and the third in the posterior portion of the superior parietal. Cutaneous and muscular sensibility and stereognostic conception probably all have some representation on the mesal aspect of the cerebrum, but we are only concerned with this indirectly in the present study of operable cerebral lesions.

The positive focal symptoms of parietal cortical and cortico-subcortical disease are impairment, usually moderate, of cutaneous sensibility, and especially of the pain and temperature senses, diminution or loss of muscular sensibility and astereognosis. When other symptoms are present they are commonly due to pressure or destructive invasion of environing regions with different functions. These may for example, if the tumor advances in a cephalic direction, be motor paresis and Jacksonian spasm with their usual accompaniments as regards the reflexes. Jacksonian spasm, however, as elsewhere indicated, occurs only rarely. If the growth invades posteriorly, the added symptoms may be visual aphasias, such as word blindness and letter blindness, and if inferiorly word deafness or other auditory aphasic phenomena. Any considerable implication of the cerebral subcortex posteriorly may give rise to hemianopsia.

If the tumor is strictly limited to the parietal lobe and does not cause any considerable pressure on the pyramidal system, both the cutaneous and the deep reflexes may be impaired in the direction of diminution. Not only is the Babinski response absent, but the cutaneous plantar response may be as negative as that which is observed in hysterical anesthesias. The knee-jerk and other tendon and muscle phenomena on the sides of the lesion remain normal, and on the opposite side either normal or somewhat depressed. If either by pressure or by destructive process, as the growth increases, the motor zone or the pyramidal tracts be involved, persistent ankle clonus and the Babinski response will appear. I have seen and recorded several cases in which the syndrome as regards the reflexes has changed during the progress of the case, as thus indicated.

To emphasize the most important features of the diagnostic symptomatology of tumors of the parietal lobe, it is necessary to briefly discuss the question of its subdivisions into different forms of sensorial and memorial representation. As I have long taught, the representation of cutaneous sensibility in all its forms is, generally speaking, located in such a manner as to surround the region of motor representation. As the motor zone according to our latest views is situated precentrally on the lateral aspect of the hemisphere, and also precentrally in the superior half of the paracentral lobule on its mesal aspect, cutaneous sensibility is in all probability represented in the postcentral convolution and in the lower half of the paracentral. As to how far the gyrus fornicatus takes part in this representation is not quite clear; not improbably the calloso-marginal fissure and the upper border of the gyrus fornicatus are concerned in cutaneous or muscular sensory representation, or in both. Cortical sensory representation is probably divided into segments for different cutaneous areas of the body, and these have topographical relations with centres and subareas of the motor region. The representation of cutaneous sensibility in the face, for example, is probably situated postcentrally and on a level with motor facial representation, that is, in the lower third of the postcentral and the adjoining anterior portion of the inferior parietal convolution, or the latter may be the seat of muscular representation for the face. The representation of cutaneous sensibility in the upper extremity occupies the postcentral convolution over a large area, extending from the facial representation to that of the trunk, this being subdivided for different parts of the upper limb. The representation of sensation of the trunk occupies a narrow strip like its correlated precentral motor centre, while sensation in the lower extremity, the anus and vagina is represented close to the longitudinal fissure on the lateral aspect of the hemisphere and more largely in the posterior and inferior portions of the paracentral lobule and the adjoining limbic lobe. Just where, in the general sensory field, the different forms of cutaneous and of muscular sensibility have their separate cortical seats it is impossible, with our present knowledge, to say.

The views here expressed with regard to the subdivisions of the cortical sensory areas, to which should be added in this connection the area for stereognostic conception, have been held by the writer for some years. They were published in the *Proceedings of the Philadelphia County Medical Society* for 1904, and also presented in a paper before the American Neurological Association in the same year. Cases of cortical lesion have occasionally been published showing the restriction of disorders of sensation and of motion to one extremity or to limited portions of an extremity or the face, but the inferences drawn have usually been based on the view that the motor region was both postcentral and precentral.

In the well-known case of Starr and McCosh,[1] a limited postparietal lesion produced loss of muscular sense confined to the opposite arm and forearm without paralysis.

Spiller[2] has recently reported a case which indicates that sensation may be affected from a cerebral cortical lesion without motor paralysis, and that the sensory alteration may be confined to one limb. A few cases similar to those of Starr and McCosh and of Spiller have been recorded.

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[1] M. A. Starr and A. J. McCosh, Amer. Journ. of the Med. Sciences, Phila., 1894, vol. cviii.

[2] Wm. G. Spiller, Journ. of Nerv. and Ment. Disease, Feb., 1906, vol. xxxiii.

The writer holds in the first place that the cortical representation of cutaneous and muscular sensation is separate from that of movement; secondly, that it is distinct not only for face, arm, trunk, and leg, but also that special segments or areas of the skin are definitely correlated with subdivisions of the sensorial and stereognostic cortex; moreover, that the restricted centres and subareas for sensation and stereognosis are correlated anatomically and functionally with special cortical motor centres. In the periphery, segments of the skin are over or closely related topographically to the muscles producing movements which are represented in the motor cortex by centres which are connected with cortical sensory centres representing these cutaneous segments or areas. The segment of the skin covering the muscles producing adduction of the thumb, for instance, is represented in the cortex by a centre which is distinct but neuronally associated with the cortical centre for this movement of the thumb.

The parietal symptom complex as stated includes astereognosis, or as some prefer to call it, stereoagnosis, the recognition of the nature and use of objects by manipulation. This is not a sense like that of pain, contact, or temperature; neither is it entirely correct to speak of the function as stereognostic perception. Stereognosis is a conceptual process. The ability to recognize objects by touching and handling them so as to obtain an idea of their form is brought about by the recalling of memorial images obtained in the first place, through such senses as contact, pain, temperature, spacing, location and position, but although thus obtained the process of recognizing objects in this way becomes an independent one. The cutaneous and muscular sensory processes are primary; stereognosis is secondary and higher. The former are comparable to what takes place in the primary visual centres in the cuneus and calcarine cortex;

the latter to the higher visual processes represented in the lateral occipitotemporal region. The stereognostic centre or area should therefore be situated in the concrete memory field or posterior association area of Flechsig. This is just where clinicopathological observation has placed it, namely, in the superior parietal convolution and the adjoining cortical expanse on the mesal plane of the hemisphere. This conceptual area, it will be seen, is surrounded on all sides by cutaneous, muscular and visual centres which administer to it. It is probable that in the process of stereognostic conception vision often plays a part; in other words in handling objects not only are cutaneo-muscular memorial images recalled, but also to some extent the visual concept of the object appears in consciousness.

A tumor rarely originates in and is still more rarely confined to the postcentral convolution. It is much more likely to have its primary nidus in the motor zone or in the postparietal region. It is for this among other reasons that ataxia and astereognosis are commonly more prominent manifestations in tumors of the parietal lobe than disorders of cutaneous sensibility. It is also likely that cutaneous sensibility has more or less bilateral representation in each hemisphere, so that destruction of both the right and the left postcentral gyres is necessary in order that decided cutaneous anesthesia shall be present.

With regard to tumors of the centrum ovale of the parietal lobe, of which I have seen several interesting examples, the differentiation between these and cortical neoplasms can be summarized in the statement that the disorders of cutaneous and muscular sensibility and of stereognostic conception are more persistent and complete in accordance with the depth of the destructive lesions, and with the additional statement that important invasion symptoms such as paralysis and tonic spasticity on the one hand, and hemianopsia and visual aphasias on the other, are much more likely to be present early in the subcortical growth.

In a case reported by Oppenheim[1] a man twenty-three years old had headache and vomiting with attacks of unconsciousness, after one of which he was left with paralysis of the left arm and left leg. The ophthalmoscope showed no optic neuritis. Pupils were normal and ocular movements were also normal. Examination showed that the ordinary use of the left arm was not much diminished, but hand pressure was slightly diminished. Tactile sense in the fingers and on the palmar surface of the hand was slightly diminished, as was also the sense of position. Disturbances of stereognostic conception were very pronounced in the left hand as tested with watch keys, steel pens and other objects. Sensations of pain and temperature were intact. In the left arm was a moderate but distinct ataxia. The left leg was dragged slightly in walking. The tendon reflexes on the left side in the leg were exaggerated to the extent even of a clonus. The left leg showed distinct ataxia. Hemianopsia was not present. Oppenheim's provisional diagnosis was tumor of the right parietal lobe reaching to the border line of the postcentral convolution and the first temporal convolution. The tumor was successfully removed. It was probably from description largely either cortical or cortico-subcortical.

In 1905 Dr. Chas. H. Frazier operated on a case of gummatous meningitis with involvement of the cortex of the parietal lobe, a case which illustrates the principles of diagnosis for tumors or other irritative lesions of this region. The patient was under the care of Dr. Wm. G. Spiller, and had previously been operated on at the Philadelphia Polyclinic by Dr. John B. Roberts. For a time he was in the wards of the writer in the Philadelphia General Hospital. The case will be fully reported by Dr. T. H. Weisenburg at the meeting of the American Neurological Association in June, 1906. The main symptoms were diminution of cutaneous and loss of muscular sensibility, astereognosis and hemiparesis, and in addition the patient had frequently recurring attacks of Jacksonian epilepsy. The spasms began with a flexor movement of the fingers and adduction of the thumb, and then spread to the forearm, arm and shoulder, sometimes involving the face and abdominal muscles and very rarely the lower extremity. This case was interesting both as indicating the location of the areas for stereognostic conception, cutaneous and muscular sensibility and movements, and also because of the peculiar manner in which the impairment or loss of muscular sense and stereognostic conception was limited to portions of the hand.

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[1] Oppenheim, Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1900, 3d vol.

In November, 1899, Dr. Keen operated for me on a case which illustrated the principles of diagnosis applicable in cases of tumor of the parietal lobe. The localizing symptoms were, in the main, impairment of cutaneous sensibility, loss of muscular sense, ataxia, astereognosis, paresis and ultimate paralysis. The patient had some verbal amnesia and fatigue on reading. At one examination he showed a temporary partial right hemianopsia. Reversals of the color fields and contraction of the fields for form similar to those supposed to be typical of hysteria were present at several of the examinations. The deep reflexes were exaggerated on the affected side and ankle clonus was present at a relatively early period after the recognition of the existence of a tumor. The Babinski response was not present at first; in fact, the metatarsophalangeal reflex was absent for a long time. Later the Babinski response appeared. Operation was performed with the view of uncovering the superior parietal convolution as the central point of procedure. The tumor was removed and the patient lived in comparative comfort for about five years, when there were signs of recurrence of the growth. Operation was again performed by Dr. Keen and a tumor at the site of the former lesion, but invading more anteriorly, was revealed and removed. The patient, however, succumbed to the shock of the operation.

In another case, a boy eight years old, operated upon by

Dr. Keen, the chief symptoms were impairment of the muscular sense, ataxia, astereognosis and paresis. Cutaneous sensibility was unimpaired or but slightly affected, with the exception that he could not locate skin sensations accurately. The deep reflexes were all exaggerated on the affected side, ankle clonus and the Babinski reflex being present.

A tumor was found and removed by Dr. Keen, it being located in the main in the superior parietal convolution (parietal of Wilder).

In another case the patient early developed marked sensory symptoms. When examination was made, three weeks before operation, it was found that the pressure sense was absent all over the hand, that the pain and temperature senses were absent in the upper extremity, most markedly in some portions of it; that the Babinski reflex could not be obtained in the great toe, and the right arm-jerks were absent. Two weeks later the patient had a severe attack of tonic spasm in the right arm, which lasted fifteen minutes. Paralysis of the limbs of the right side, in a few days, was practically complete; no hemianopsia was present. The profound disturbances of sensation in this case were probably due to early implication of the parietal subcortex; the motor symptoms to subsequent invasion of the motor subcortex. Operation and necropsy showed that the tumor had invaded both subcortex and cortex of the parietal region and the subcortex of the postfrontal region; it had evidently originated in the subcortex, as it was just breaking through the parietal cortex at the time of operation. Operation in this case was performed by Dr. W. W. Keen.

In still another case recorded by me which was operated by Dr. W. J. Hearn, the first focal symptom was loss of power in the right leg. At the time of examination loss of power was marked in leg and arm, more complete in the latter, and some facial paresis was also present. Knee-jerk and patellar clonus were present on the right, although, strange to say, both the ankle clonus and the Babinski reflex were absent. The right leg exhibited spasticity and tremor and the right arm was also spastic. Sensation to touch and pain was nearly lost in the right upper extremity; it was but slightly impaired in the right lower extremity. Impairment of muscular sense was also a marked feature. Astereognosis was present and became more and more positive as the case progressed to its termination. All forms of cutaneous sensibility and muscular sensibility were tested by the usual methods, with the result of showing impairment, which as time passed became more and more complete in the upper extremity. The so-called senses of locality, position, pressure and spacing were found wanting. In the right lower extremity the quadriceps-jerk and knee-jerk were exaggerated, and patellar clonus was marked. The focal symptoms became more and more profound, but did not alter except in degree. The patient suffered from headache of great severity and optic neuritis, which passed into atrophy. The diagnosis was made of a dense tumor of large size, probably in the main subcortical, situated chiefly in the parietal region, possibly invading the motor region and extending or compressing the posterior limb of the internal capsule and the optic radiations. The position of the growth was also determined by a Roentgen ray investigation, the shadow being very decided. In the operation performed by Dr. Hearn a portion of the tumor was removed.

Although this case eventually became cortical by its growth from within outward, it presented the characteristic features of a subcortical postfrontal and parietal growth—namely, early and marked paralysis, tonic spasticity without Jacksonian convulsions, loss of all forms of sensation and astereognosis; the reflexes, as might be expected, were of uncertain diagnostic importance because both the motor and the sensory subcortex were involved.

In a case of large parieto-occipital tumor the mistake of operating for the motor region was made. The patient was seen late, when the motor symptoms overshadowed all others, and one or two points in the past history of the patient were not obtained until after the operation, especially some facts which seemed to indicate that hemianopsia had been present. At the time when it was decided to operate, the patient's symptoms were such as to make it impossible to determine clearly the existence of symptoms like hemianopsia, astereognosis and impairment of cutaneous and muscular sensibility. Because of the predominance of motor symptoms, such as paralysis with contracture and exaggerated reflexes, it was decided to operate for the motor region, although the position of the growth was regarded as somewhat uncertain.

The general symptoms of brain tumor were present. The operation was performed by Dr. A. C. Wood, of Philadelphia. The patient died several weeks after the operation, and it was found at necropsy that the posterior line of the opening was just anterior to the large tumor mass.

TUMORS OF THE REGION OF THE JUNCTION OF THE PARIETAL, OCCIPITAL AND TEMPORAL LOBES, THE HIGHER VISUO-AUDITORY REGION.

The inferior parietal or subparietal convolution curves around the posterior extremity of the Sylvian fissure to unite with the first or supertemporal convolution, and in like manner another portion of the subparietal convolution curves around the posterior extremity of the first temporal fissure to join with the hinder portion of the second temporal convolution. Two angular or curving convolutions are thus formed. To that which is constituted by the convolution which winds around the posterior extremity of the first temporal fissure the term *pli courbe* or angular convolution has long been applied. The general region formed by the curving and uniting extremities of these parietal and temporal convolutions is one of the highest functional importance. It is the visuo-auditory area of the cerebral zone of speech. In the angular convolution, and perhaps extending somewhat beyond it posteriorly, are situated the centres for word seeing, letter seeing and number seeing. The primary auditory centre, that area the destruction of which on both sides will cause cerebral deafness to primary sounds, is situated in the retroinsular convolutions and the coterminus part of the posterior extremity of the first temporal convolution. The centres for word hearing and for other higher forms of auditory representation, as for natural musical notes and intonation, are situated on the outskirts of the primary region of auditory representation. The word hearing centre has been placed by me at the junction of the first and second temporal convolutions in their posterior parts. The centre for intonation is probably situated more anteriorly in the first or second temporal convolutions. With regard to the function of word hearing and of intonation, the representation is much more evolved in the left than in the right hemisphere.

A tumor destroying the angular or highest visual region will cause word blindness, letter blindness and number blindness, or some of these; also verbal amnesia, visual agraphia, paragraphia, alexia, dyslexia and paralexia. The chief invasion symptoms will be auditory aphasias and hemianopsia if the disease advances in a postero-inferior direction, or if in an antero-superior direction, astereognosis and disorders of cutaneous and muscular sensibility.

A tumor occasionally originates in the first or second temporal convolutions, of which I have seen instances. If such a growth originates in this portion of the left hemisphere the central feature of the syndrome will be word deafness with its aphasic accompaniments, such as paraphasia, paralexia and word dumbness. Commonly some visual aphasia is associated with the auditory aphasia because of the proximity of the higher visual centres and their associations with the higher auditory centres.

Dr. Stanley Barnes[1] has reported an interesting case which illustrates the principles of focal diagnosis in a case of tumor situated so as to destroy or partly destroy the fibres which connect the visual and auditory centres together and these centres with the speech and graphic centres in the third and second frontal convolutions. A tumor was present which had evidently grown very slowly over a period of nine or ten years. The growth was bounded in front by the postcentral, and behind by the angular convolution, which was somewhat involved by young growth; above the superior parietal lobule was also somewhat implicated. It would seem from the description that the tumor occupied largely the inferior parietal convolution, although the recorder of the case speaks of it as bounded below by the supermarginal convolution. The patient had a series of attacks extending over many years. In these he was dazed or dizzy, had shimmerings of light before the eyes, and had numbness beginning in the right hand and extending to the arm, side and leg. Other symptoms were word dumbness, paraphasia, concentric contraction of the visual field on the side opposite to the lesion, and at times slowness in understanding what was said. Spontaneous writing, writing from dictation and from copy were all markedly affected. He had an imperfect right hemianopsia. He was not object blind, and had no loss of power or impairment of sensation or astereognosis. After the early seizures, the focal symptoms disappeared almost entirely, but were more persistent as time went on between the attacks. Optic neuritis was not present until late. Headache, vertigo and nausea and vomiting were among the general symptoms. The growth would probably have been largely included in the upper portion of the opening advised for the exposure of the visuo-auditory centre, and in the lower portion of the parietal opening. By moving the visuo-auditory opening a little forward and toward the mesal line the tumor would have been well uncovered.

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[1] Stanley Barnes, Review of Neurology and Psychiatry, vol. i. 1903.

MIDDLE AND INFERIOR TEMPORAL REGIONS.

Tumors are infrequent in the middle and inferior temporal regions, which include the lower half of the second and the whole of the third, fourth and fifth temporal convolutions. The uncinate and hippocampal convolutions are of course too centrally situated to be accessible to the surgeon. A growth involving the second, third and fourth temporal convolutions might be removed, at least in part, but in the case of the third and fourth temporal convolutions, not without some destruction of the brain substance around the tumor. I have seen a case of mid-temporal tumor which at an early stage, probably a year before the death of the patient, might have been removed by a careful surgical procedure. Such growths, like tumors in other situations, may arise in the membranes of the middle fossa and gradually compress and invade the temporal lobe. The symptoms produced by a tumor in the mid-temporal region are not well known. In one case studied by me they included word dumbness, hemianopsia and a partial mind blindness and word blindness. The tumor in this case, however, at the time of investigation just before death had invaded considerably toward the occipital lobe. A tumor strictly limited to the mid-temporal region will probably give as its main symptom word dumbness because of the destruction of the naming centre in this region, presuming its existence here. If such a centre should not here exist, as has been asserted, the same symptoms will be produced by destruction of the tracts passing through the various sensory centres on the receptive side of the brain to the motor speech centre in Broca's convolution.

The opening used in the effort to reach the middle and lower temporal regions should be made as low as possible on the side of the skull, its superior limit being on a line which would correspond with the horizontal branch of the Sylvian fissure just before it turns upward posteriorly.

TUMORS OF THE OCCIPITAL LOBE, THE LOWER AND HIGHER VISUAL AREAS.

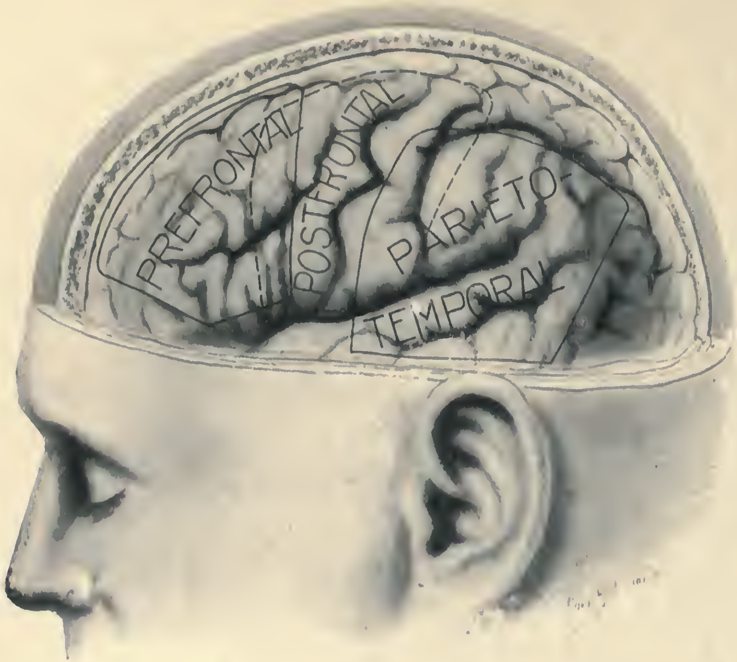
The primary cortical visual centres, the centres of the optic perception field, are situated entirely or almost entirely on the mesal surface of the occipital lobe, the centres of color recognition being probably situated more ventrally on its tentorial aspect. The secondary or higher visual area, however, covers a large extent of the lateral occipito-temporal expanse. Centres of object recognition in its various forms are situated from the first to the fifth occipital convolutions inclusively and occupy also the contiguous caudal extremities of the adjoining temporal convolutions. These centres of object recognition, as has been indicated by me in other contributions, have probably a considerable subdivision, but the subareas or centres partly or wholly authenticated are few in number. They include the centres for word seeing, letter seeing and number seeing in the angulo-occipital region and centres for persons, places and natural objects in the rest of the lateral occipital lobe. I have suggested also the probable existence of centres for geometric and architectonic forms close to the representation of the symbols of language, but no facts have been forthcoming as yet substantiating this suggestion.

It is evident from a study of the anatomy of the skull and brain that only those occipital tumors are operable, or at least can be operated upon with much hope of success, which are situated wholly or largely in the lateral occipital region. It is true that the mesal surface of the lobe and even its tentorial surface may be to some extent explored, and by a good chance a small tumor may be shelled out of either of these regions. Another point worthy of recollection in this connection is that a large part of the occipital lobe may be removed, as was done in one of my cases by Dr. Frazier, without fatal or even serious symptoms, except of course the necessary loss of vision; so that a tumor situated in any part of the occipital lobe may be worthy of the consideration of the surgeon.

A tumor of the lateral occipital or occipito-temporal region, the area of higher visual representation, if the lesion be a left-sided one, will give for its main symptoms word, letter and number blindness, complete or partial, and if partial their accompanying aphasic defects such as paralexia, agraphia and paragraphia, optic aphasia or word dumbness, and in some cases mind blindness, which in unilateral lesions is usually incomplete and transient or paroxysmal. As tumors, even those which originate in the membranes, usually sooner or later involve the subcortex, hemianopsia is often present. Other compression and invasion symptoms are chiefly auditory symptoms, such as word deafness and paraphasia, although ataxia and astereognosis may appear if the growth advances forward. In addition to the hemianopsia, if its advance is subcortical, hemianesthesia and hemiparesis or paralysis may appear.

The Wernicke reaction is absent. The deep and superficial reflexes in the extremities are usually not changed. Motor affections and psychic disturbances except those concerned with visual concrete memories are absent.

In the last section I have spoken of the manner in which the highest visual and the highest auditory areas could be exposed jointly or separately. If the symptoms are almost purely visual the osteoplastic opening should be more posterior than those referred to in that section; in other words, it should be made so as to include the entire lateral aspect of the occipital lobe with adjoining extremities of the parietal and upper temporal convolutions, as indicated by the space marked occipital in Fig. 2. Practically the region referred to in this figure would be exposed by an opening one side of which would be parallel with the longitudinal fissure, about one-half inch from the mesal line, and the other parallel with the lateral sinus about one-half inch from the cranial line corresponding to this sinus. The other two



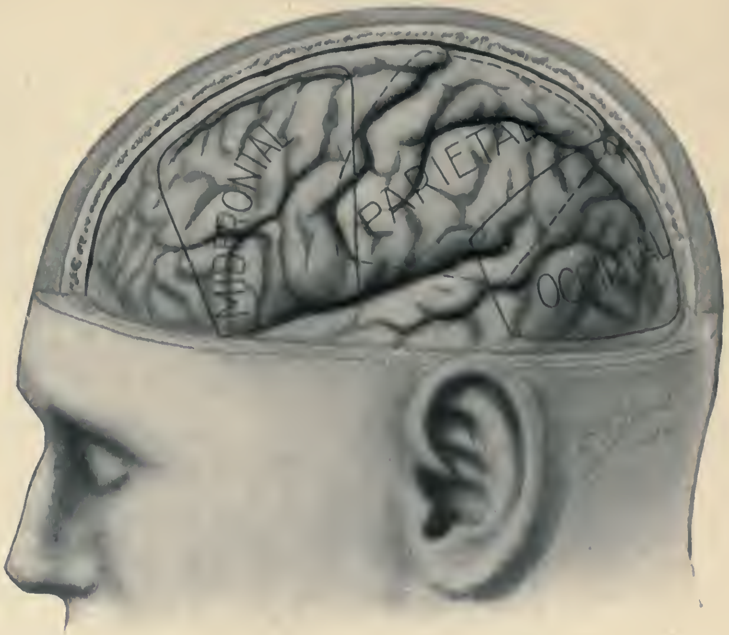


FIG. 1.—Diagram showing the convolutions uncovered by standard osteoplastic openings for the prefrontal, postfrontal (motor), and parietotemporal (visuoauditory) regions.

FIG. 2.—Diagram showing the convolutions uncovered by standard osteoplastic openings for the midfrontal, parietal, and occipital regions.

sides of the opening could be made in the most convenient manner possible.

I have seen no cases of successful operation on the occipital lobe, although in two cases of occipital tumor operation was performed for me by Dr. Frazier. The growths, however, were not reached, probably because they were located tentorially or meso-tentorially. In one case the patient died, but no necropsy was allowed; the other patient is still living and in fair general health, although a portion of his occipital lobe was cut away several years since. This case is one of those referred to by Drs. Spiller and Frazier in their paper on " Decompression."

A tumor of the right basal occipito-temporal region, if the optic radiations and cuneus are not involved, is very difficult of diagnosis. In a case of this kind occurring in my practice, Dr. Frazier performed a subtentorial operation. This patient had the well-marked general symptoms of brain tumor, including optic neuritis of an extremely high grade. Hemianopsia, as tested by the hand, which was the only method which could be adopted owing to his relative blindness, was not present. The only symptoms which seemed to have any localizing value were a drooping of both eyelids and nystagmus on lateral movements. Associated upward movements were performed promptly, but the patient was unable to keep the balls upward, and convergence was very slightly performed. The tumor in this case was large and dense, and had destroyed the occipito-temporal region in large part.

GENERAL REMARKS ON SOME OF THE SOURCES OF ERROR IN THE FOCAL DIAGNOSIS OF BRAIN TUMOR.

In spite of the numerous advances, some of them quite recently made, in our knowledge of focal physiology and focal diagnosis, mistakes are still made by those whose business it is to locate tumors or other operable lesions for the surgeon, although it is notable that these mistakes are growing fewer. At several points in the preceding pages some of the sources of error in focal diagnosis of brain tumors have been pointed out, as, when speaking of the occurrence of cerebellar, sixth nerve and other symptoms because of the tilting of the brain, and its jamming into the foramen magnum by a prefrontal growth, as recorded in one of the cases of Collier; as also when referring to the fact that Jacksonian epilepsy may be simply a part of the convulsive attack in a case of idiopathic epilepsy, and that this form of spasm, due to a variety of focal lesions and general states, may be attributed to the presence of a neoplasm in the motor region; and again, as when speaking with references to illustrative cases, of the masking of parietal or prefrontal symptoms by the late and more obtrusive motor manifestations due to pressure and invasion.

The paper of Collier on the false localizing signs of intracranial tumor, to which reference has already been made several times, presents in an admirable way the manner in which focal symptoms in cases of brain tumor may mislead the neurologist and surgeon. Almost every point to which he calls attention has been personally observed by me, and to some of the points I have called attention incidentally in various papers. The contribution of Collier has, however, particular value because of the succinct and eminently practical manner in which he has marshalled these false portents, and in the remarks which follow I shall again avail myself of his observations.

The interpretation of symptoms due to pressure exerted upon a functional area or tract as the evidences of tumor impairing or destroying this area or tract and the obscuring of the early and more important symptoms by those which are caused by late invasion or encroachment are matters upon which emphasis has already been made, but because of their importance it will be of service to refer to them again in this general discussion of diagnostic errors, especially as the neurologist is sometimes called late in the course of a case of brain tumor to decide as to the site and operability of the lesion. He may see the patient for the first time after the disease has been present, recognized or unrecognized, for months or even years. The patient at this time may present a striking symptom-complex. It may be that of hemiparesis or hemiplegia, with occasional attacks of Jacksonian epilepsy; it may be hemianopsia with some attendant sensory phenomena; or it may be aphasia or agraphia; or other symptoms formerly present may be overshadowed or obscured, as for instance, in a case of hemiplegia such clinical phenomena as hypæsthesia, ataxia and astereognosis, or in a similar case, psychical, aphasic and agraphic phenomena. It follows, therefore, that when the localization of a growth is for the purpose of exactly fixing the limits of an operation, the method in which the tumor has spread, as determined by a careful study of the history of the development of symptoms in the case, is often of the greatest importance. Probably the mistake most frequently made is that of supposing a tumor originating and largely occupying the prefrontal lobe, and only encroaching upon the graphic, speech or motor region, chiefly occupies the last.

Compression symptoms usually precede for a short time at least those of absolute encroachment, and this fact needs to be borne in mind. It may be said, however, that symptoms due solely to pressure are rarely absolutely continuous and can be differentiated by a close study of the case. In many cases the pressure seems to transiently let up or recede, probably because, in some cases at least, of the filling and emptying of the cysts which are often connected with tumors.

Collier's discussion of the disappearance of true localizing signs in late stages of brain tumor is in part a discussion of the effects of invasion. Among other things, he shows how the late symptoms may obscure entirely the presence of a cerebellar growth.

One must be always on his guard not to be misled in his efforts at accurate localization because of the presence of abducens palsy. Different theories in explanation of the frequent occurrence of this symptom have been given. Probably it has most often been attributed to the effects of pressure upon the nerve in its long intracranial course, and this doubtless accounts for it in some instances. Collier has suggested another explanation which is reasonable and may explain the symptom in some cases. He has shown that as the result of pressure of a large prefrontal tumor, the brain may be more or less tilted so that the cerebellum and oblongata are jammed into the foramen magnum. One of the consequences of this compression and tilting of the entire brain is traction on nerves like the sixth and third, which have a long caudocephalic course. The sixth nerve especially would be subject to such traction; in other words, the case would afford an illustration of indirect traumatic traction palsy. The nerves like the fifth, seventh, eighth and others which run transversely or more obliquely are not subjected to the same amount of traction. Whatever may be the correct explanation, the diagnostician should always bear in mind that while paresis or paralysis of the sixth nerve may be of strict localizing value, that is, may indicate involvement of this nerve, it may on the other hand be an indirect symptom of tumor of large size situated in various regions of the brain.

Fifth nerve symptoms, either sensory or motor, may be present in a case of brain tumor in which this nerve is not directly involved. In two cases referred to by Collier, in which the tumor was situated in the left prefrontal region and frontal centrum ovale respectively, fifth nerve anesthesia and hemianopsia were both present. Collier attributes the symptoms referable to this nerve to pressure exerted on the middle fossa by a large prefrontal tumor on the same side.

The occurrence of bilateral spastic paresis in a case of brain tumor is not infrequent and may be misleading. In one of my cases of tumor of the cerebello-ponto-oblongatal recess this symptom was most marked, and hydrocephalus of considerable degree was found on necropsy. So marked was the spastic paralysis in this case that I suspected some form of spinal disease, as meningomyelitis, in addition to the brain tumor which was evidently present from the general, as well as the local, symptoms. Collier lays much stress on the occurrence of this symptom of bilateral spastic paresis due to hydrocephalus accompanying a brain tumor in various intracranial situations. A tumor of the brain stem is most likely to give rise to this affection. Affections of various cranial nerves may be associated with the bilateral spastic paresis.

In discussing errors in focal diagnosis, the question of multiple tumors needs brief consideration. When more than one growth is present in the cranium, it is rare to find these growths in the cerebrum proper. Usually one will be present somewhere in the cerebrum, and another or others at the base or in the cerebellum. In multiple sarcomatosis, growths may, for instance, be present in both cerebellopontile recesses, in the middle and anterior fossa and even in various positions in the spinal canal. In very rare cases similar tumors may be present at nearly corresponding points in both hemispheres. A tumor of the callosum occasionally invades in both directions. In bilateral tumors, if recognized, operation on both sides might be considered; in other cases the growths are inaccessible and inoperable.

Our concern is with the operable tumors of the cerebrum, but a few words might be said about the diagnosis of some of those intracranial neoplasms which are inaccessible and inoperable, especially as in a few instances these give symptoms which may seem at some stage to point to accessible regions. Tumors of the thalamus give inco-ordination and anæsthesia, and according to some authorities central pain. If the pulvinar or optic radiations are involved hemianopsia will be present. When the superior cerebellar peduncles are included in the lesion hemichorea and hemiathetosis may be present, and when the pregeniculum is implicated the Wernicke pupillary reaction. Forced laughing or crying have also been noted as thalamic symptoms. Monoplegias or hemiplegias described in connection with thalamic lesions are probably due to the destruction of the neighboring parts of the internal capsule. A tumor involving the thalamus and posterior part of the posterior limb of the internal capsule might at some stage of its development have some of the appearances of a tumor of the parietal subcortex.

Tumors of the lenticula and caudatum give symptoms which cannot with our present knowledge be well separated from growths affecting the internal capsule. These symptoms are mainly hemiparesis or hemiplegia with disorders of speech, and if the lesion is in the caudal portion of the lenticula, hemianæsthesia may be present. Von Bechterew[1] has described a symptom of lesion of the lenticula designated by him as pseudomelia paræsthetica. To a patient suffering from this affection the limbs appear to be in a position in which they are not.

With regard to tumors of the chiasm and the pituitary body, attention should be concentrated upon bitemporal hemianopsia, olfactory disorders and symptoms indicative of implication of the third, fourth and sixth nerves. The part which some believe is played by the lesions of the pituitary body in the causation of acromegaly should be borne in mind. In tumors of the crura, symptoms of third nerve involvement on one or both sides and paresis of the extremities on one or both sides are prominent. Chiasmic, pituitary or crural lesions can scarcely be mistaken for any form of operable lesion of the cerebral cortex or subcortex.

Tumors involving the cinerea of the region of the Sylvian aqueduct, the brain stem or the cranial nerves at the base anywhere in their course give pseudobulbar, nuclear and cranial nerve symptoms—a statement sufficient to indicate the method of their separation from operable cerebral lesions.

The symptoms of tumors of the pons will vary according to the exact location and extensions of the growth. Among them the most notable are alternating hemiplegias of different type, paresis or paralysis of separate or of associated ocular movements, dysphagia, anæsthesia, which may be of the crossed type, astereognosis in rare instances, painful affections of the trigeminus and vasomotor and thermic changes.

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[1] Von Bechterew, Neurol. Centralbl., No. 17, September 1, 1905.

The chief symptoms of tumors of the quadrigeminum are impaired sight or hearing or both, paralysis of ocular movements, bilateral or associated, and inco-ordination especially in attempts to walk or stand. Other symptoms have also been recorded, such as nystagmus, automatic repetition of words, and a tendency to move backward or other forced movements. Focal symptoms referable to neighboring parts, such as the cerebellum and the ascending and descending tracts in the pons oblongata may be present and the general symptoms of a neoplasm, such as headache, optic neuritis, vertigo, nausea and vomiting, are usually marked.

As tumors of the cerebellum and the cerebellopontile angle have been discussed elsewhere by the writer and by his colleagues in the preparation of the present series of papers, attention will be directed to cerebellar growths only in the briefest possible manner. It should be remembered that an accessible parietal tumor may give ataxia as one of its prominent manifestations, but that a growth giving this symptom is to be differentiated from a cerebellar growth, causing ataxia by the fact that the ataxia is more likely to be confined to the extremities of one side, and especially to one upper extremity in parietal lobe cases, and that in the same cases, astereognosis and impairment or loss of cutaneous and muscular sensibility are usually present, these being absent in a cerebellar neoplasm. I would refer elsewhere, however, for a full consideration of this subject.[1]

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[1] Tumors of the Cerebellum, New York Medical Journal and the Philadelphia Medical Journal, February 11 and February 18, 1905; reprinted with papers by Drs. Frazier, Weisenburg, de Schweinitz and Ludholz as a monograph.

With regard to the focal diagnosis of brain tumors from such lesions as abscess, hemorrhage and acute softening, from embolism or thrombosis, little need be said. The principles of diagnosis are the same as in the case of tumors. The differences in symptomatology depend chiefly upon the differences in the nature of the lesions, although in the case of hemorrhage and acute softening, the rapidity of development is of some assistance in differentiation. An abscess may cause either more or less pressure than a tumor; probably when entirely confined within the brain substance, less rather than more. An abscess may be extracerebral that is, of the membranes, as for instance when in the middle fossa. In such cases the symptoms will not only be indicative of the physiological area disturbed or injured, but also of dural irritation, and it may be of disturbance of the functions of cranial nerves. The fact that an intracerebral abscess is often latent for a considerable or even a long time needs to be borne in mind. After all the differentiation between abscess and tumor will depend largely upon the history of probable causation, and upon the presence or absence of the indications of sepsis. In the presence of purulent otitis or of some other source of pus, abscess becomes more probable, and yet the diagnosis of abscess must not be reached simply in this way, as I have known not a few instances in which brain tumor was present in cases with the evidences of recent or old aural disease.

With regard to the diagnosis of hemorrhage from tumor the usually sudden occurrence of hemorrhage is an important point; it may also develop more or less slowly. It must be kept in mind that a hemorrhage sometimes takes place during the growth of a tumor, either into its substance or on its borders. I have seen instances of such hemorrhages in cases of gliomata and even of gummata. A point of value in the differentiation of hemorrhage from tumor is that in some cases of brain tumor, although the neoplasm has been long existent, the onset of focal symptoms of diagnostic importance is rapid. This is probably to be accounted for by the brain ceasing after a time to accommodate itself to the new-growth, and also by the sudden or rapid increase of its destructive influence on vascular regions invaded by it. Acute softening from embolism is always sudden, and embolic apoplectiform symptoms are of a special type. The presence of endocardial and valvular lesions or of other pathological sources of emboli will of course have great weight in diagnosis. In softening from thrombosis, which may be sudden or rapid in onset, although the changes in the vessels leading up to the attack may have been long present and increasing, the absence of the general symptoms of brain tumor and of the pressure and irritation symptoms are important.

The diagnosis, focal and general, of brain tumor from general paresis is sometimes difficult; it is especially so in cases of prefrontal tumor. In several instances I have hesitated and postponed operation because of this difficulty. In certain cases of general paresis, as is well known, the symptoms are of a quiet or apathetic type. The patient does not exhibit the extravagant optimism of the typical paretic. In a few cases most of the decisive spinal and cranial physical symptoms, such as changed knee-jerks, ataxia, and disturbances in the iritic reflexes, may be absent. The scene is dominated by dementia showing itself in loss of memory, disorientation, impaired judgment and lack of self-control. The speech disorders are never entirely absent, but unfortunately in the case of a prefrontal and midfrontal growth these may be present. In such a case the general symptoms of brain tumor if present are of the greatest value, but one or even all of them may be absent and yet a neoplasm be present. It is sometimes necessary to wait in such cases for the development of aphasia and agraphia if the tumor is left sided, or for Jacksonian epilepsy and paretic symptoms if right sided.

Extracted from the American Journal of the Medical Sciences, May, 1906

FOUR CASES OF DOUBLE HEMIPLEGIA (SYPHILITIC ENCEPHALITIS, CEREBROSPINAL SYPHILIS), WITH AUTOPSY.[1]

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The following four cases of double hemiplegia illustrate some of the pathological changes which may occur in the nervous system as a result of syphilis. The pathological conditions commonly met with in cerebral syphilis are: gummatous formation; meningitis; disease of the bloodvessels; and areas of softening, usually the result of vascular disease. Less frequently syphilitic encephalitis occurs, although this is undoubtedly more common than some believe.

The first case (from the service of Dr. Wiliam G. Spiller, at the Philadelphia General Hospital) illustrates several of these processes, causing an infrequent train of symptoms, and is further interesting because an exact pathological diagnosis would have been impossible without a careful microscopic study of the specimens.

Case I.—A woman aged fifty-four years, a domestic by occupation, was admitted to the Philadelphia General Hospital, April 2, 1901, suffering from right-sided hemiplegia.

Her father died of old age and her mother of phthisis. She had had scarlatina and pertussis when a child, and in the winter of 1901 she had an attack of pneumonia. Three months prior to her admission she was suddenly seized with unconsciousness, lasting ten minutes. No paralysis followed this attack, but a few weeks later, upon arising in the morning, she complained of dimness of vision and difficulty in expressing herself. A week later, and a month before she was admitted to the hospital, the right arm became weak and numb, and the right leg was slightly weaker than the left. There was no palsy, but the tongue protruded to the right.

When examined a week after her admission there was decided weakness of the whole right side—arm, hand, and leg—and she dragged her right foot when walking. The tongue was protruded to the right, and she had some difficulty in expressing herself. She used the wrong words, and miscalled the names of objects, though after making several attempts she would finally recall the proper word. She would call a pocket-knife a "screw-knife," and a pen a "pencil to ink with." There was slight hyperæsthesia on the right side below the knee, and sensation was blunted in the right arm. The right arm and leg were both spastic.

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[1] Read before the Philadelphia Neurological Society, April 5, 1905.

All the reflexes were exaggerated. The knee-jerks and the biceps-jerks on both sides were increased, but more so on the right than on the left side. The Babinski phenomenon could not be obtained on either side at this time. Her lungs and abdomen showed no abnormalities. The pulse was slow and rather weak, but regular.

On April 14 she fell twice, without apparent cause, and without disturbance of consciousness. After the last fall the left arm was found to be weak and spastic. The pupils were equal and responded to light, and there was no hæmianopsia. There was no sensory change. The Babinski reflex was present on both sides. Three days later the grasp was nil on the left, and she lifted her arm with difficulty. The knee-jerks were increased on each side, but more on the right. The left leg was at this time weaker than the right. There was no abnormality of the heart and lungs.

On April 27, ten days later, she fell again, and upon assisting her to her feet it was found that there was some loss of power in the legs, that she could not articulate distinctly, and that the face was flushed. The right leg was spastic and the knee-jerks were increased. The Babinski reflex was present, but there was no clonus.

She was examined by Dr. William G. Spiller, July 6th, and the following conditions were found: Both feet were contractured in the position of talipes equinovarus. The contractures, however, could be overcome by passive movements, but were more marked on the right side than on the left. When the legs were elevated and allowed to drop they fell as if paralyzed. In the few days preceding the examination there had been incontinence of urine. There was no marked atrophy.

The knee-jerk on the right side was prompt, but not exaggerated, and on the left it was absent, and no movement of the quadriceps muscle could be seen. The Achilles jerk on both sides was absent. Babinski's reflex was present on both sides to a marked degree.

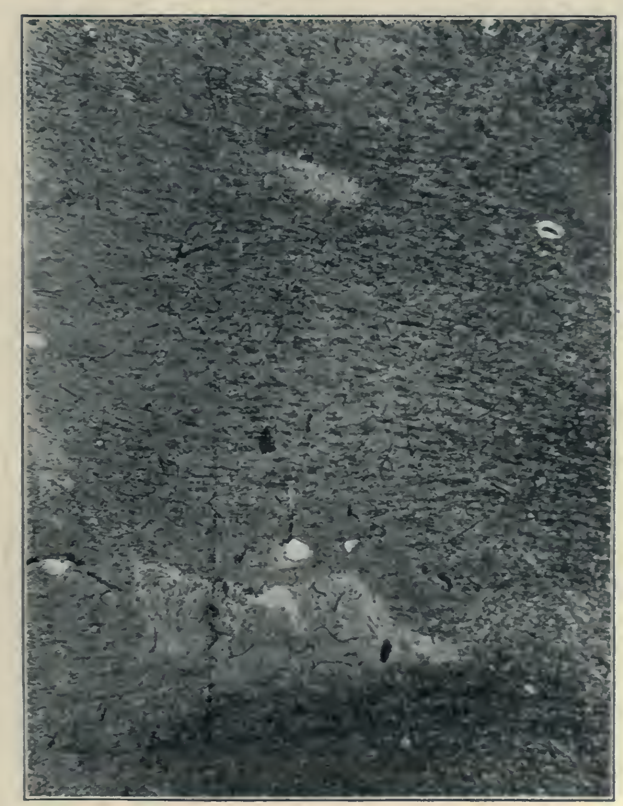
When stuck with a pin the patient made no response, excepting when the sole of the foot was tested.

The right arm was in the position of an old hemiplegia. The fingers were slightly flexed, especially toward the ulnar side. The hand was extended at the wrist, and the forearm was flexed to a right angle on the arm. This contracture could be overcome by passive movements.

The biceps-jerk on the left was slightly exaggerated, while the olecranon and wrist-jerks could not be obtained. The arm was spastic, and when allowed to fall showed increased tonicity.

The face and head were drawn to the right. There was marked conjugate deviation of the eyeballs. The pupils were equal and the eyelids could be closed. When the lid of the right eye was irritated the eyelid closed firmly. When the left lid was irritated the lid was closed less firmly than on the right side. There was no puffing of either cheek indicative of facial palsy. The patient died of pneumonia.

FIG. 1



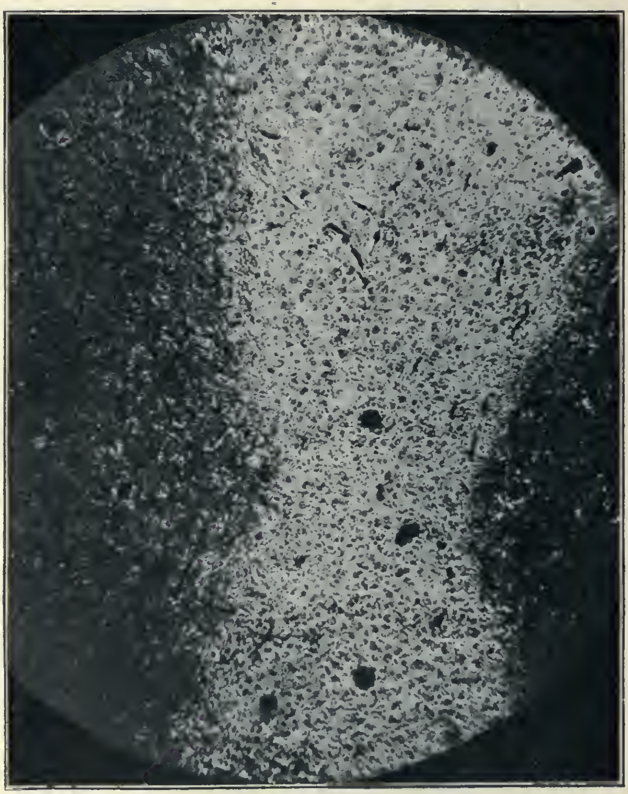
Showing two microscopic areas of softening in the cortex.

The pathological diagnosis was lobar pneumonia; chronic nephritis; cirrhosis of the liver, and arterial sclerosis.

The brain and spinal cord were placed in Müller's fluid, and sections from various portions were stained by the Weigert hæmatoxylin method, and by hæmalum and acid fuchsin.

In the right paracentral region there was a pronounced meningitis. The cells of Betz were distinctly diseased, the bodies of the cells being swollen and the nucleus displaced. In the cortex, just below the surface, were seen several small areas of softening, consisting of masses of fatty granular cells, closely packed together. (See Figs. 1 and 2.) In places these fatty, granular cells extended within the tissue, along the outer layer of the cortex just below the surface, without forming any distinct area of softening. Elsewhere in the cortex in the paracentral region there existed much round-cell infiltration, around the vessels especially, and elsewhere more moderately. The perivascular spaces were distended, and everywhere in this region the bloodvessels were markedly congested. A few small hemorrhages were present. In the right occipital region a round-cell infiltration of the pia was observed. No conspicuous change was noted in the cortex.

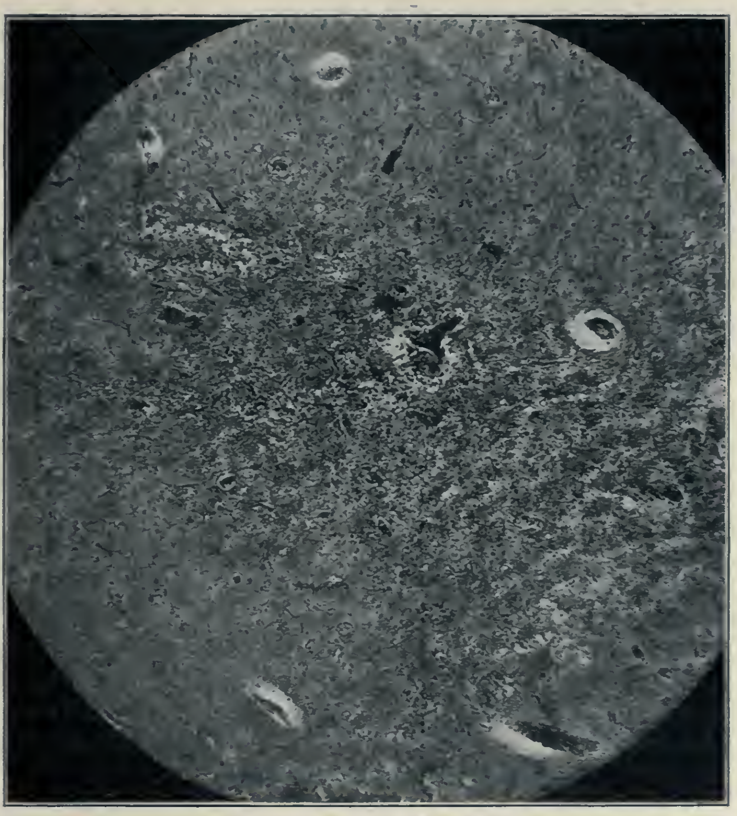
FIG. 2



Area of softening in the cortex showing the fatty granular cells. (Higher magnifications of areas shown in Fig. 1.)

In the right frontal region the pia was intensely infiltrated. The cells consisted mainly of mononuclear round cells, but a few large cells were found containing two or three nuclei. The pia was much thickened, and there were, in places, newly formed bloodvessels. The vessels of the pia showed distinct change. In some of these the disease was more intense in the adventitia, and the intima, while showing some proliferation, was less affected. In others all the coats of the vessels were thickened, and the proliferation of the intima was so intense that the lumen was almost obliterated. One large vessel, the walls of which were thickened, was obliterated entirely, save for four small lumens around which the exudate was more deeply stained, suggesting the formation of denser tissue in these places. In the cortex, just below the surface corresponding to the site of an intense meningeal process, the nerve tissue was loose, and the normal appearance

FIG. 3



Area of inflammatory encephalitis; distribution of perivascular spaces; round-cell infiltration; congested vessels.

could not be made out. Small holes were seen here and there, probably due to resorption of the fibres, or the result of œdema. A little deeper, in the white matter, a focus of inflammation was observed. Here there was intense, round-cell infiltration, and the vessels were much congested. This area was more or less circumscribed, and the tissue around it was undamaged. (See Fig. 3.)

Elsewhere in the cortex distention of the perivascular spaces was present, and the vessels were generally congested. An intense meningitis was present around the optic commissure, and the bloodvessels within the commissures were much congested. Here, also, were found enlarged perivascular spaces and round-cell infiltration. No degeneration of the fibres of the optic commissure was discovered. No degenerated fibres could be seen in the pyramidal tracts of the pons, stained by the Weigert method, but when stained by the Marchi method quite a considerable degree of acute degeneration was seen on one side.

The pia over the cerebral peduncles was the seat of an intense, round-cell infiltration, especially marked around the vessels. A similar condition was also found in the pia of the medulla oblongata. By the Weigert stain no degeneration of the peduncles was observed. In the pons there was an area of softening $\frac{1}{8}$ x $\frac{1}{4}$ inch in area, just above the median fillet.

In the medulla oblongata the left anterior pyramid took the Weigert stain a little less deeply than the right. Considerable acute degeneration was seen, however, in the sections stained by the Marchi method. The right crossed pyramidal tract and the left direct tract in the cord showed, by the Weigert method, a slight degeneration.

Summary of the Pathological Findings. There was present a round-cell infiltration of the pia, with disease of the bloodvessel walls, especially marked in the frontal region, but present at the base and convexity as well. In the paracentral cortex small areas of softening were present, and in the frontal region there were areas of inflammatory encephalitis. The cortical vessels were congested, and round-cell infiltration and distention of the perivascular spaces were common.

The cells of Betz were diseased. In the pons and the medulla oblongata recent degeneration of the nerve fibres was seen, and in the medulla oblongata degenerated fibres were discovered by the Weigert method.

The bloodvessel change calls for some discussion, as the findings were not entirely characteristic of the description of syphilitic arteritis as laid down by various authorities. Some of the vessels showed a preponderance of the disease in the adventitia and media, while in others all the coats of the vessels were greatly involved, the lumen of the vessel being frequently almost entirely or quite obliterated by the proliferation. In some parts of the brain, the occipital region principally, the vessels showed little change. But wherever the meningeal process was intense the bloodvessels were much diseased.

Heubner[13] claims that syphilitic disease of the vessels originates between the endothelium and the membrana fenestrata. Round cells escape later from the vasa nutritia of the vessels, giving rise to the granulation tissue of the intima. According to Friedländer,[10] the process is an endarteritis obliterans. On the contrary, Baumgarten[4] believes that the primary alteration arises in the adventitia and its vascular apparatus, causing secondary thickening of the intima. Baumgarten[5] has described disease of the bloodvessels in syphilis, consisting of gummatous deposits between the adventitia and the media, and in both of these coats of the vessel resembling in structure a meningeal node. He claims that occasionally the deposit within the vessel directly communicates with these nodes. He found instances in which there existed periarteritis and mesarteritis without proliferation of the endothelium. Oppenheim[25] reported a case also in which the disease of the vessel originated directly from a gummatous tumor. On the other hand, the vessels may alone be diseased, as Marchand and others have described. Rumpf looks upon the change in the intima as a reactive proliferation, having its origin in the vasavasorum. Köster[16] also is of the opinion that the origin of the process is in the vasavasorum of the adventitia, and that the inflammation of the muscularis follows with the escape of lymphoid cells, giving rise to a mesarteritis, and, finally, an endarteritis. Oppenheim[27] believes that in the majority of cases the specific meningitis attacks the vessels directly, and may involve one or all of the coats.

A study of the change in the vessels found in my case sheds little light upon this mooted question, though the evidence may point to the conclusion that the process sometimes involves primarily the outer coats, when it would seem to be a part of the pathological change in the meninges, or that the intima may be primarily attacked, the inflammation arising from direct irritation of the poison circulating in the blood.

The cases of syphilitic encephalitis in the literature, with a few exceptions, are so lacking in the description of the finer histological changes that satisfactory deductions cannot be made.

Cases reported as syphilitic encephalitis are often merely examples of cerebral softening; others lack microscopic description, and are therefore valueless; while still a very few other references are found to what may be termed a true inflammatory encephalitis.

As illustrating the first group of cases, the case of Obermeier may be cited. In his case softening of the cortex was present beneath gummatous inflammation of the meninges. Griesinger[11] collected a number of cases in which foci of softening were present in the brain, probably of vascular origin. He stated that the apoplectic form of brain disease in syphilis was in the main due to encephalitic foci, and that hemiplegia was often the result.

Other cases have been recorded by Tournier,[34] Mildner,[22] von Bechterew,[3] Lancereaux,[18] and Rosenthal,[30] while Nonne,[24] Ljunggren,[19] Gowers,[12] and Naunyn[25] mention softening in the brain as a frequent occurrence in brain syphilis. According to Oppenheim,[27] in the majority of cases the brain softening originated in obliterated vessels, or in consequence of a thrombosis. Again, softening has occurred around gummas, while specific meningitis has given rise in some cases to the same condition.

I could find no case except that of Barrett[2] in which the post-mortem findings were comparable to the small areas of softening in the cortex in my case, most of the cases reported being examples of extensive foci of softening, readily seen by the naked eye. The cortical areas of softening I have described are remarkable in that they are detectable only with the microscope. But in most of the cases of softening reported microscopic descriptions are lacking.

Encephalitis of inflammatory origin in syphilis is a less common, but by no means a rare, occurrence. Gowers[12] regarded inflammation of the brain of specific origin, however, as rare, and while admitting that primary softening and induration had been met with, there was always the question, are they not of vascular origin. They are, he says, seldom specific when inflammatory. In Barrett's case of syphilitic encephalitis the findings were in many respects similar to those found in my case. There were present, in his case, an inflammation of the pia-arachnoid, and proliferation and degeneration of the vessels. In the vessels the intima was most frequently affected, while the adventitia was more involved than the media. The nerve cells were degenerated, and proliferation and degeneration of the glia and vessels of the cortex were present. There were also areas of encephalitis, composed of granulation tissue, besides foci of simple necrosis related to a neighboring vessel. Besides these a few gummatous foci and some hemorrhages were found.

Oppenheim[28] reported a case of softening independent of blood-vessel change. Two foci were observed in the brain, showing the presence of round-cell infiltration, plasma cells, "körnchenzellen," destruction of nerve tissue, and new vessel formation. He compared this to the encephalitis of Hayem and Friedmann. In a case reported by Charcot and Goumbalt[7] two foci were seen in the medulla oblongata, containing a number of cells which appeared to be highly developed neuroglia cells. There seemed to be a proliferation of the neuroglia cells, which, pressing against each other, ceased to be properly nourished, and degenerated in consequence.

In Obermeier's[26] case there was a proliferative and degenerative process in the pia, and in the cortex the pia cells besides being changed in form were degenerated. The neuroglia was proliferated, and there was a round-cell accumulation. Elsewhere the perivascular spaces were distended, and areas of circumscribed infiltration were seen. The bloodvessels were intensely diseased, and some of them were obliterated.

Alzheimer[1] reported a case in which the cortex was the seat of a high-grade infiltration. The vessels were narrowed or obliterated; perivascular infiltration was present, and the nerve cells were altered. There was, in a case of Möller,[21] inflammatory infiltration of the dura, and besides thrombosis of the artery of Sylvius there was found "sclerosis" and degenerative change in the basal ganglia.

Cases of encephalitis (so reported), with less satisfactory descriptions, are those of Tugendreich,[33] in a child one month old, Schaffer,[32] Jastrowitz,[14] and Köbner.[17] Heubner[13] collected from the literature up to 1877, one hundred and fifty cases of simple inflammation of the brain of syphilitic origin, of which he found only twenty-four trustworthy cases.

In connection with the study of the foregoing case of double hemiplegia, an opportunity to study three other cases, in the Philadelphia General Hospital, was offered by the courtesy of Dr. William G. Spillier.

Case II.—G. S., aged sixty-nine years. Family history negative. He denied specific infection, and gave a history of vertigo for two years previous to admission. On the day before admission (September 16, 1904) he had a dizzy attack and fell, although he was not unconscious. Dizziness and headache followed. Since that time he has not been able to walk. When examined later (the notes do not say exactly when) the left side of the body was spastic, especially the leg; the reflexes were generally exaggerated, although they were not distinctly elicited on the left side on account of the spasticity. Babinski's sign was observed on both sides. He urinated involuntarily, and complained of shooting pains. There was also some tenderness over the nerve trunks. Three months later (December 22, 1904) after "feeling badly" he found he was unable to feed himself with the right hand. Distinct weakness of the right arm and leg was noticed, associated with some difficulty in articulation. The speech was rather explosive, although no true aphasia was demonstrated. The grasp on the right side was weak, and the muscular power of the arm was lowered. The reflexes were prompt in the right arm, while in the right leg the knee-jerk was exaggerated. No patellar or ankle clonus could be elicited. Babinski's sign was prompt on both sides.

There was no anæsthesia or paræsthesia present. The tongue was protruded to the left, and the pupils were normal and responded to light and in accommodation.

When examined on March 22, 1905, I found the patient in the following condition: He was bedfast, and unable to move the arms or legs. When spoken to he made no sign that he heard, nor did he obey commands. He would not protrude his tongue, and made no effort to move his arms or legs, or to wrinkle his brow. However, he chewed his food, and the act of deglutition appeared to be unaffected. The arms and legs were held in marked flexure contractures, equally on both sides. The arm-jerks were exaggerated, but the leg reflexes could not be elicited on account of the extreme contractures. The arms were œdematous from the elbow downward, and over the flexor and extensor surfaces of the elbows there were blisters, evidently trophic in origin. On the heel of the right foot, and the first metatarsal joint on the left, there was an area presenting a black appearance, and resembling local gangrene. There was no discharge or ulceration, and a similar lesion was cured previously, simply by the application of dry antiseptic dressings. There was incontinence of urine and feces.

The patient died of interstitial nephritis and cirrhosis of the liver a few days later. The brain and spinal cord were hardened in formalin, and sections from the various regions of the brain, pons, medulla, and cord were stained by the Weigert and Marchi methods and by hæmalum and carmine.

The pia of the paracentral lobule was slightly infiltrated with cells, and the bloodvessels were thickened. A slight perivascular round-cell infiltration was present in the cortex. In the frontal and occipital regions similar changes were found.

There was an area of softening in the pyramidal tract on one side of the pons, and the pia over the pons was the seat of a round-cell infiltration, and its vessels were thickened. The fibres of the pyramidal tract, near the area of softening, stained fairly well, except those which protruded upon the softened area. On the other side the fibres of the pyramidal tract were stained deeply. In the medulla on one side the pyramidal fibres were stained less deeply than on the other side. Here also the pia showed round-cell infiltration.

In the cervical region of the cord each crossed pyramidal and direct pyramidal tract stained by the Marchi method appeared much degenerated. This was also shown by the Weigert method.

A moderate, round-cell infiltration only was found in the pia of the cervical region of the cord. Amyloid bodies were present in large numbers in the posterior columns. In the lumbar region the crossed pyramidal tracts were slightly degenerated. The pia was infiltrated with round cells, but not intensely so, while within the cord a mild degree of perivascular infiltration was observed. The Marchi method showed a recent degeneration of the crossed pyramidal tracts. The microscopic findings were those of cerebrospinal syphilis. It should be noted here that the history of specific infection was denied.

Case III.—C. B., aged fifty-seven years, a spinner by occupation, was admitted to the Philadelphia General Hospital, February 6, 1905. Her father is living and well. Her mother died of "heart failure." Two brothers are living and well. Two brothers died of phthisis, and one sister died suddenly.

She had borne eleven children, all of whom died in infancy. She never had any miscarriages. She suffered the ordinary diseases of childhood, and had always been temperate.

A year before her admission to the hospital there was a history of paralysis of the right arm and leg, from which she largely recovered. During the last week in January, 1905, three weeks prior to her admission, she suddenly lost power in the left arm and leg, without disturbance of consciousness. Since this time there had been no return of the power, in fact she gradually grew weaker.

When examined by myself March 20, 1905, she presented the following conditions: There was no facial palsy. The tongue was protruded straight. The grasps were both feeble, but the left more so. The left arm was spastic at the elbow and shoulder, while the right showed no tendency whatever to spasticity. The right leg was slightly spastic, and there were beginning contractures in the foot. Voluntary power in the right arm and leg was present only in a moderate degree. The left leg was held up across the body in a flexed position, the knee reaching the right side of the body. This contracture could be only partially overcome, and effort to do so was attended by pain. The arm-jerks on the right were unobtainable on account of the contractures present; on the left side they were exaggerated. The knee-jerk on the right side was exaggerated; on the left it could not be obtained, due to the contractures. The ankle-jerk was present on both sides, but no clonus could be developed either on the right or left. Babinski's phenomenon was present equally on both sides. There was no local atrophy discoverable anywhere. The urine and feces were passed incontinently. Sensation was intact. Examination of her lungs disclosed no marked abnormality.

The patient died April 4, 1905, of chronic nephritis, and at the autopsy a tumor of the brain was found involving the right frontal lobe anteriorly, and the median fissure was slightly displaced to the left.

The tumor measured about three centimetres in all directions. Sections from this tumor presented the characteristic appearance of a gumma. Sections from the several regions of the brain and from the medulla were stained by the Weigert hæmatoxylin method, and with hæmalum and acid fuchsin. The pia in the right and left paracentral regions was infiltrated with round cells, and some of the bloodvessels were thickened. In the cortex in these regions there was also some infiltration around the bloodvessels, and in the cortex below the diseased pia. A similar condition was found in the right and left occipital regions, but it was less marked. In the occipital region the cortex did not show anything abnormal. The pia covering the peduncles was infiltrated with round-cells, but the peduncles themselves were not pathological. There was a small area of softening in the left pyramidal tract of the pons, situated near the raphe, and the pia covering the pons was the seat of a round-cell infiltration. In the medulla the pyramid on the left side took the Weigert stain less intensely than on the right side, and the pia covering the medulla was moderately infiltrated with round cells. In the cervical, thoracic, and lumbar regions on the left the crossed pyramidal tract was degenerated. This was not very marked, and could be more readily seen by the naked eye than with the microscope. The cells in the lumbar and cervical regions were normal. The pia of the cord at the levels studied was thickened and infiltrated with round cells.

The pathological changes in this case were characteristic of cerebrospinal syphilis.

Case IV.—S. D., aged fifty-two years, a widow, was admitted to the Nervous wards of the Philadelphia General Hospital, December 29, 1904. The family history was negative. She had suffered from all the ordinary diseases of childhood, and had had smallpox at the age of forty. Her health had always been good otherwise. She had one child, born at term, which died in twenty-four hours. She never had a miscarriage. For years she had been a steady, hard drinker.

A year ago she suddenly became unconscious, and upon awakening she was weak in the left arm and leg. From this she recovered largely, although there was some defect on that side. A few days before her admission she fell to the ground, was unconscious, and afterward was found to be weak on the right side.

When examined by myself, February 13, 1905, she was generally emaciated. She moved the right side of her face less readily than the left, although the forehead was wrinkled equally well on both sides. The tongue was protruded straight, and the speech was somewhat indistinct, but there was no aphasia. The pupils were equal and responded normally. Both arms were weak, and while the grasps were about equal, the movements of the left arm were more restricted than those of the right. There was distinct loss of power in both legs, in the left more than the right. She was unable to stand. Both arms and legs were distinctly spastic, and on the left side there were contractures at the shoulder, elbow, hip, and knee. These could be overcome with a moderate amount of force. The first two toes of the foot were hyperextended.

The reflexes were everywhere increased, and the elbow-jerk and biceps-jerk much exaggerated, and equally on both sides. The knee-jerks were large and spastic, and a rectus clonus could be developed on the right side. There was no ankle clonus, and the ankle-jerk was absent. Babinski's sign was present on the left side, but not on the right side, although at a previous examination by Dr. William G. Spiller he noted the presence of this reflex on both sides. There was much muscular soreness in the legs, not localized to the nerve trunks. A pinprick was felt distinctly all over the body. There was no local atrophy.

The patient died April 20, 1905. At the autopsy an excess of fluid escaped when the skull was opened, and the pia and arachnoid were œdematous, while the basilar vessels were thickened. On the right side there was a large area of softening involving the temporosphenoidal lobe. On the left side a small area of softening was observed in the occipital lobe. Unfortunately the brain was largely destroyed at the autopsy, and portions of the cortex from the occipital and temporal regions, and the pons and medulla and cord only, were saved for microscopic study. In the sections from the temporal region there existed a round-cell infiltration of the pia, and around the bloodvessels, extending into the cortex. The same condition was present in the sections from the occipital lobe. No degeneration was present in the cerebral peduncles, but the pia covering them was infiltrated with round cells, and the vessels at the base of the brain were much thickened. In the medulla, stained by the Weigert method, one of the anterior pyramids was slightly paler than the other one, while the other pyramid appeared to be normal. The pia and bloodvessels of the medulla presented the same condition as that found in the pia of the peduncles. In the cervical region there was some slight degeneration of both crossed pyramidal tracts, which was more marked on one side than on the other. The pia of the cord at this level was greatly infiltrated with round cells. Some perivascular round-cell infiltration was observed within the medulla and spinal cord. The nerve cells of the anterior horns of the cervical region were in good condition, although the thionin stain was not employed. In the lumbar region the crossed pyramidal tracts were slightly degenerated. The nerve cells of the anterior horns appeared to be normal. The pia at this level was also infiltrated with round cells. The lesion in this case was also that of cerebrospinal syphilis.

In two of these cases the paralysis of the opposite side followed the primary attack of hemiplegia in a year's time; in one, after three months; and in the other, six weeks. In all of the cases the characteristic microscopic appearances of cerebrospinal syphilis were seen.

After searching the literature rather carefully for cases of double hemiplegia, I was surprised to find very few references, and these rather unimportant, and adding but little to our stock of knowledge on this subject. It may be that such cases are not reported under the title of double hemiplegia, for they are not of very uncommon occurrence.

Jolly[15] cited an interesting case of hemiplegia of sudden onset followed later by paralysis in both legs, the left leg remaining palsied to the end, some motion still remaining on the right side. Sensory symptoms were present. At the autopsy a pure myelitic focus was found in the spinal cord in the upper thoracic region, giving rise to secondary degeneration.

A case is reported by Brown-Séquard of double hemiplegia, and one in the *Medical and Surgical Practice*,[23] September 18, 1869, the symptoms of which are not important enough to quote.

In one case, reported by Dawson,[8] a boy of four became suddenly paralyzed in the right arm, leg, and tongue. Later, as the paralysis was improved, a gradual rigidity and loss of control of the left arm came on. The brain was softened, especially the cerebellum. Further details of the postmortem examination were lacking.

Dunn[9] described a case in which there was right-sided hemiplegia, hemianæsthesia, aphasia, and right lateral hemianopsia, following an attack of unconsciousness. The symptoms improved greatly in ten days, and in three weeks the patient was as well as before the attack. Two years later, upon awakening in the morning, he was found to be paralyzed on the right side. There was partial hemiplegia, anæsthesia, and absolute blindness. The palsy improved to the normal in four weeks, but the sight remained greatly impaired.

Köbner[17] recorded a case, in a man, of right-sided paralysis of sudden onset. Later there was transient palsy of the left arm, and repeated attacks of paralysis on the right side, arm, leg and face. At the autopsy there was found a syphilitic endarteritis, meningitis, and chronic encephalitis. There was a gumma in the pons, and secondary degeneration of the pyramids on the left side. The left lenticular nucleus was the site of encephalitis and a cyst. Areas of softening were also found in the pons.

CONCLUSIONS. 1. In brain syphilis small microscopic foci of softening may occur in the cortex, the result probably of cutting off of the blood supply, and giving rise to hemiplegic symptoms and mental disturbances.

2. Syphilitic inflammatory encephalitis, while described comparatively rarely, may occur, and is probably due to inflammation originating in the small vessels of the cortex by the syphilitic poison circulating in them.

3. The change in the bloodvessels is not always uniform. Sometimes the adventitia is more intensely involved and sometimes the intima.

4. Double hemiplegia, if not uncommon, is certainly not frequently described.

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LESIONS OF THE LEFT FIRST TEMPORAL CONVOLUTION IN RELATION TO SENSORY APHASIA.

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THE centre for word-hearing in right-handed persons is generally believed to be in the posterior part of the left first temporal convolution and possibly of the second. According to Bastian, the merit of having determined this region belongs to Wernicke, but Bastian thinks that disturbance (Bastian, " Aphasia and Other Speech Defects." H. K. Lewis, London, 1898) to a very marked extent of the functions of the visual word centre as a result of a lesion in the upper temporal convolutions is not so universal as Wernicke and Dejerine believed. Thus out of sixteen recorded cases of sensory aphasia in which the lesion was pretty closely limited to the hinder part of the first, and more or less of the second temporal convolutions, in only five is any mention of some amount of word-blindness. In the sixteen cases to which Bastian referred, motor aphasia existed in six, some paraphasia in six, aphasia and paraphasia in one, and voluntary speech seemed to be, but to a less extent, affected in three.

Von Monakow says that so many instances of word-deafness, resulting from a lesion of the left first temporal convolution in right-handed persons are recorded, that the relation between this lesion and the word-deafness cannot be doubted. There are cases of lesion confined to the left second and third temporal convolutions with sensory aphasia, and others with a lesion confined to the posterior part of the left first temporal convolution without aphasia, and in illustration of the latter statement he refers to Byrom Bramwell's Case 10, *Brain*, 1899 (Von Monakow, " Gehirnpathologie," 2nd edition, p. 929).

Still, word-deafness with destruction of the posterior part of the left first and second temporal convolutions is not so common that new cases may be ignored, and the two following cases are reported to form a contrast with the third, in which destruction of the left first temporal convolution in a right-handed man did not cause word-deafness.

Case I. William Jones, aged 29 years, was admitted to the Polyclinic Hospital, to the service of Dr Steinbach, May 12, 1901, with the diagnosis of fractured skull. He was struck in the left temporal region with a base-ball bat. When he was admitted he had profuse hæmorrhage from the left ear, and this continued most of the night. He has been unconscious since admission, moves his limbs somewhat, and has tried to get out of bed, but does not seem to know when anyone is speaking to him. He is on liquid diet, and only a small amount can be forced into him. The bowels have been opened by enema. The temperature is $100\frac{1}{5}°$; pulse, 60 ; respiration, 14. Hæmorrhage from the ear has entirely ceased, and he lies in a semicomatose state most of the time.

May 17.—Paralysis, both motor and sensory, has appeared in the right limbs. The pulse is slow, and more feeble than it was previously. He is still unconscious, but less restless than before.

May 18.—He seems to understand some things that are said to him, but he cannot do more than occasionally mutter a reply. There is slight motor power in the right leg. The left eye is inflamed, the right iris responds to light.

May 20.—Paralysis in the right leg is disappearing. He tries to answer questions, but cannot make himself understood.

May 22.—He sings occasionally, but cannot make himself understood; seems to understand some things said to him.

May 24.—His right arm moved to-day; he has twitchings of the right angle of the mouth, otherwise his condition is about the same.

May 26.—He has regained some use of his right arm.

June 3.—A slight twitching of the mouth is noticed on the right side. He speaks when spoken to, but replies by meaningless words.

July 6.—*Notes by Dr Spiller.*—The tic of the right side of face in its lower portion still continues, and probably is caused by a large scar on the right side of the neck, extending from the middle of the clavicle to the mastoid process. The paralysis on the right side of the body has almost entirely disappeared. His speech is as follows: "Well, sir, I know I use to have good. I can ever do ever know. Yes I have been plenty; I have been impose; I know where I at, who I not; I would to work to work plenty plenty." This is mingled with words that cannot be understood. All this was in answer to the question of his name. He does not recognise his name when it is mentioned among other names. When told to sit up in bed he did so; when told to lie down, he did so; when told to give his hand he did not seem to understand; he sat up a second time on command, when all gestures were avoided. He was probably word-blind.

June 15.—An operation over the posterior part of the left first temporal convolution was advised. Dr Steinbach made a horse-shoe shaped incision (base downward) over the temporal region, dissected the periosteum from the bone, exposing a stellate fracture; and at the superior border of the squamous portion of the temporal bone a depression was seen. The patient was trephined just above the point of depression, and the opening was enlarged by rongeur forceps to about the size of half a dollar. The dura was found torn and the brain substance protruding. The finger, when inserted, entered a large cavity filled with degenerated brain material and blood-clots.

After removal of this broken-down tissue, the cavity was irrigated with normal salt solution, and a silk thread was inserted as a drain. At this stage hæmorrhage started, and the cavity was packed with iodoform gauze. The scalp wound was now brought together and sutured.

June 16.—The patient had a very comfortable day. He does not show any signs of improvement as yet.

June 17.—He still remains word-deaf. No weakness of the right side is detected. General condition is good.

June 24.—The wound was re-dressed to-day, and all the packing was removed. The wound is in an excellent condition. The patient is allowed to be up.

July 1.—The wound of the scalp has healed nicely, but there is no improvement in the mental condition of the patient. He was sent to the Philadelphia General Hospital, July 5th, 1901, and came into the service of Dr Spiller.

His word-deafness persisted unchanged. He occasionally understood a word here and there in a sentence, and guessed at the meaning of the rest of the sentence. After he had been in the nervous wards about a year, he became insane and was referred to the insane department.

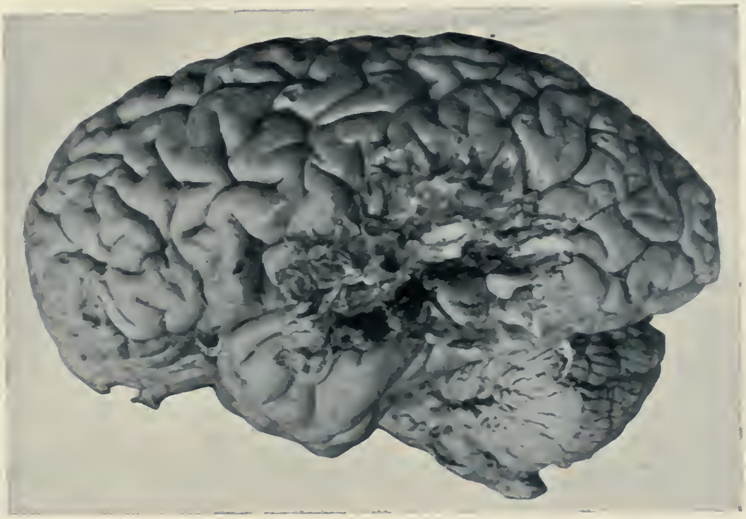
July 17, 1902.—He says he hears people talking to him, hears the devil and thinks he saw him also, that he jumps at him and wants to kill him.

July 19, 1902.—He no longer has auditory hallucinations, but has been unmanageable at times, and very abusive and violent, has refused to remain in bed, and has wandered about the grounds, and threatened the nurse several times.

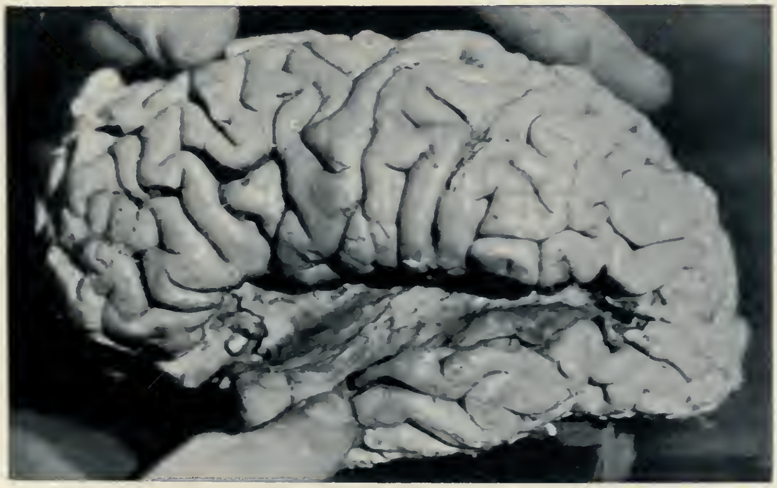
August 25, 1902.—He has delusions of persecution, and has again shown evidence of hallucinations of hearing, which, with the visual hallucinations, are especially interesting on account of the injury of the centre for word-hearing and of the optic radiations.

I am indebted to Dr Charles W. Burr for the brain of this patient. An area of sclerosis implicates the posterior part of the first and second left temporal convolutions and lower part of the parietal lobe. It extends to the posterior part of the island of Reil, and to the optic radiations, and may implicate the latter.

The history in the second case is very brief. The man was in the service of the late Dr Frederick A. Packard, at the Pennsylvania Hospital, and the brain was given to me by Dr Packard and Dr Simon Flexner in April 1901. The former wrote that the man had had Cheyne-Stokes breathing many days before his



Brain in Case I.



Brain in Case III.

death, and was so deaf that it was impossible to obtain any but the most meagre history from him, and the deafness had been long-standing. He had no friends, consequently his former history could not be obtained.

The greater part of the left first and second temporal convolutions are sclerotic and the cortex here is extremely atrophied.

The third case, in contrast to the other two, is very interesting. The patient was under the care of Dr Herman B. Allyn, and the clinical notes are from him. I am much indebted to Dr Allyn for them.

October 14, 1900.—F. R. S., aged 57 years, complains of painful cramps in his right upper and lower limbs. The leg is jerked up in flexion and subsequent extension is slow and painful. His clinical history is as follows :—

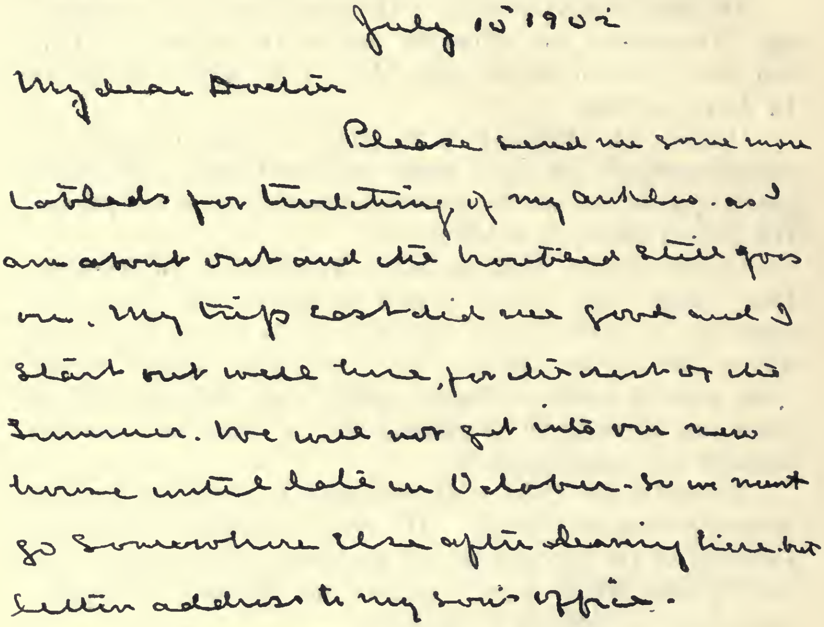
On January 28, 1881, he fell in New York in an apoplectiform attack. He recovered with disturbance of speech and some loss of power on the right side of the body. Four years ago he had another seizure. He is now partly aphasic, cannot write without making mistakes, walks with difficulty, and at times has difficulty in swallowing and in urination; also has hiccough and vomiting spells.

November 29, 1902.—His condition is about the same as at the previous examination. His chief complaint is spasmodic twitching of the leg muscles and weakness of the ankles. He has diplopia, which is not constant, and difficulty in stepping down from the sidewalk to the street.

October 21, 1903.—He cannot walk without help. Speech is no worse. He has been obliged for years to open the anus with the finger in order to start a stool.

January 7, 1904.—He is weaker and cannot stand, as his legs bend under him and he does not know where they are when he is in bed. He has involuntary micturition, and painful spasms of the right leg.

In reply to questions, Dr Allyn informed me that the patient was never musical, had always had remarkably acute hearing, and there were no words he had difficulty in understanding. He did not swing or drag his right lower limb so long as Dr Allyn had had him under his observation—about four years. He had some loss of power on the right side, and the right ankle was likely to give way under him and he was liable to fall. He always shook hands with his right hand. His gait was somewhat ataxic. He understood *all* that was said to him, but was frequently at a loss for a word in speaking, although he could carry on a conversation, tell anecdotes, and was very entertaining. When he was fatigued or was not feeling well his speech became at times muffled and indistinct, so that he would have to be asked to repeat. When he had difficulty in saying a word, he would say, with an air of embarrassment and a slight laugh, "You know what I mean." His speech was probably much worse following his first attack in 1881 than at any time since. He could write legibly, but rarely did write, and was liable to misuse words in writing, and therefore had his letters supervised by a member of the family. In the letter reproduced there are several mistakes: "address" for "addressed," "tabled" for "tablets," "troubled" for "trouble." He was sound mentally, had good judgment and reasoning powers, but his memory had failed somewhat. He read constantly. He was right-handed.



July 15 1902

My dear Doctor

Please send me some more tabled for twitching of my ankles. as I am about out and the troubled still goes on. My trip east did me good and I start out well here, for the rest of the summer. We will not get into our new house until late in October. so we must go somewhere else after leaving here. but letter address to my son's office.

Letter written by the patient (Case III.).

The microscopical examination shows that the patient had tabes. The first left temporal convolution is entirely destroyed and appears as a shrivelled mass of tissue, and the sclerosis extends to the posterior limb of the internal capsule and to the posterior horn of the lateral ventricle, and implicates the optic radiations. The second left temporal convolution is intact, and the first temporal convolution on the right side is unusually well developed.

The only explanations that are suggested to me for the preservation of word-hearing in this case are that the centre for this function was largely in the posterior part of the left second temporal convolution, or that the right first temporal convolution had been unusually well developed during the patient's entire life, and was capable of assuming the function of the destroyed left first temporal convolution.

Some cases in literature give evidence that the right cerebral hemisphere may sometimes replace the left in the functions of speech, even in adults.

Freund reports the following case :—

A woman, right-handed, 73 years of age, had apoplexy, and following this, right hemiplegia and aphasia. Improvement occurred, and most important was the condition of word-hearing. She could distinguish between sounds, she understood what was read to her from the newspaper, she repeated sentences if they were short and spoken slowly to her. She did not have alexia, letters were written correctly, and she copied correctly. In spontaneous writing, and in writing to dictation, or after long copying, repetitions were frequent. The understanding of spoken words seemed to be impaired only when she was spoken to rapidly or in too long sentences, or in unusual words. Right lateral homonymous hemianopsia and the inability to name objects seen or touched by her indicated the location of the lesion.

At the necropsy an area of softening was found in the left temporal lobe, and had caused much destruction. The lesion extended to the middle of the occipital lobe posteriorly, and to about 1½ cm. behind the anterior end of the corpus callosum anteriorly.

Freund believed that the restoration of word-hearing in this case is to be explained by the assumption that the right temporal lobe functionated in place of the destroyed left temporal lobe. Wernicke suggested a similar explanation for a case in 1874, and this view has been held by Pick, Kauders, V. Monakow, Entzian, and others. (Freund, *Neurologisches Centralblatt,* Oct. 1, 1904, p. 914, and Oct. 16, 1904, p. 965.)

In the discussion of this case, Sachs, of Breslau, expressed doubt concerning vicarious action of the right hemisphere in a person so well advanced in years, and Pick shared the doubt. Freund acknowledged that the objection was forcible, but because of the intense destruction of the left temporal lobe he assumed that the patient had since youth made partial use of her right temporal lobe in the function of word-hearing.

Bastian reports a case in which the whole of the left superior temporo-sphenoidal convolution was destroyed, with the exception of the anterior one-third, 4½ cm. in length. Of the middle temporo-sphenoidal convolution, the anterior 5 cm. were perfectly intact; but posteriorly, only a narrow portion of the inferior part of the convolution remained, and that was in a discoloured and degenerated condition. The left angular and marginal convolutions were destroyed. The patient understood what was said to him and could repeat all simple common words. His spontaneous speech was limited to his name and short affirmative or negative answers, or short phrases. He understood what he read. Bastian believed that the destruction had been gradual, and that a gradual development in the functional activities of the corresponding convolutions of the right hemisphere occurred. This case had been under observation eighteen years, and there had never been word-deafness or word-blindness. Presumably the man was right-handed. ("Aphasia and Other Speech Defects," by H. C. Bastian. H. K. Lewis, London, 1898.)

A case is reported by Jolly in which right hemiplegia and motor aphasia followed an apoplectic attack. Word-hearing for simple questions was preserved, but complex questions were not understood. Reading was impossible. The necropsy revealed destruction of Broca's convolution, and of the first and partially of the second left temporal convolutions. Word-hearing in great measure is said therefore to have been preserved, notwithstanding complete destruction of the left first temporal convolution, and Jolly thinks the right hemisphere functionated in place of the left, although the patient was 73 years old at the time of the attack, and lived but a short time after the attack began. (Jolly, *Centralblatt für Nervenheilkunde und Psychiatrie*, Oct. 21, 1899, p. 593.)

The third case that I have reported can not be explained on the ground that the patient was left-handed, as Dr Allyn is sure that he was not. There is evidence that in left-handed persons the speech centres are in the right cerebral hemisphere. In a case reported by Joffroy, the patient was word-deaf, word-blind, paraphasic and agraphic, and an area of softening was found in the middle portion of the right first temporal convolution, with meningo-encephalitis about it. Only three cases of sensory aphasia, according to Joffroy (Kussmaul, Touche, Köster), with lesions in the right side of the brain have been reported, and Joffroy's case makes the fourth. The three cases occurred in left-handed persons. Joffroy's patient wrote and ate with the right hand, and those who had known him had not observed that he was left-handed. (Joffroy, *Revue Neurologique*, Jan. 31, 1902, p. 112.) Mills and Weisenburg have reported a case of word-blindness in a right-handed man, with the lesion in the right cerebral hemisphere. (Mills and Weisenburg, *Mèdicine*, Nov. 1905.)

The case of Bloch and Bielschowsky seems to show that a lesion of the anterior part of the left first and second temporal gyri may cause word-deafness, and that involvement of the posterior part of these gyri is not always present when word-deafness exists. In their patient the hearing of sounds was excellent, but that of words was completely lost. Reading and writing were not tested. A hæmorrhagic focus was found in the point of the left temporal lobe, and it extended backwards and downwards. (Bloch and Bielschowsky, *Neurologisches Centralblatt*, Aug. 15, 1898.)

We need more cases of word-deafness from lesions of the left first and second temporal convolutions in their anterior portion before we can locate the centre for word-hearing in this part of the brain.

Probst seems to have shown that the centre for music-hearing may be in the anterior part of the first left temporal gyrus. A patient of his had motor and sensory aphasia, she could repeat only a few words, and these conveyed no meaning, but she could sing and articulate the words in singing correctly. She recognised songs with which she had been familiar, and sang them correctly after some one else, and was able to sing unknown tunes, but without the notes. (Probst, *Archiv für Psychiatrie*, vol. xxxii. No. 2, p. 387.) The anterior part of the first left temporal gyrus was not implicated in the lesion.

The explanation of the substitutional functioning of the right cerebral hemisphere applies to motor speech as well as sensory.

A case that Byrom Bramwell reports shows that acute and complete destruction of Broca's area, and of the anterior end of the left island of Reil, in a person who has always been right-handed, may produce merely a very temporary motor aphasia. Bramwell believed that in his case the cortical centre in the right hemisphere corresponding to Broca's convolution was more highly educated than it is in the great majority of right-handed persons, and was able to carry on immediately the functions of the left motor-vocal speech centre. According to his views, the right cerebral hemisphere has more to do with speech than is usually taught, and the speech centre and speech functions are bilaterally represented in the brain, but not to the same extent in each hemisphere.

A somewhat similar case has been reported by Collier, and probably other similar cases could be found in the literature. (Bramwell, *Brain*, Autumn 1898; Collier, *Lancet*, 1899.)

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June, 1906.

HYSTERIA SIMULATING BRAIN TUMOR, WITH A REPORT OF TWO CASES.[1]

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(From the Department of Neurology, University of Pennsylvania.)

It is well known that hysteria may resemble in its symptomatology almost any organic nervous disease, and that a differential diagnosis is at times most difficult. This is especially so if there really exists some organic disease, and the hysterical symptoms are superadded.

While our conception of the nature of hysteria is by no means clear, it is probable that the disease is of cerebral origin and depends upon a temporary perversion of function of the cortical or basal centres. By no other means can we explain such symptoms as hemiplegia, monoplegia, disturbances of sensation, of vision, and so forth.

It must also be remembered that in almost every organic nervous disease, indeed it may be said that in any disease of any organ or of any portion of the body, some neurasthenic or hysterical symptom may be found. Every physician encounters it daily in his practice and it is the recognition, treatment, and the cure of this that makes the successful physician.

It is a question, at times, whether it is more difficult to recognize hysterical symptoms from organic or the reverse. No better illustrations of this can be found than in the disease, multiple sclerosis. Brain tumors offer at times the same difficulty, cases being recorded in which hysteria was diagnosticated, whereas at necropsy a brain tumor was found, and others, in which the so-called organic symptoms were really hysterical. If the latter error is committed it may lead to serious results, for a needless surgical operation may be performed for brain tumor. The writer has recently had under his observation such a case, and the record of a similar case is given, for the notes of which he is indebted to Dr. Wm. G. Spiller.

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[1] Read before the Medical Society of the State of Pennsylvania, held at Scranton, September 26 and 28, 1905.

Case I.—A woman, thirty-two years of age, was sent to me by Dr. John T. Krall, of Philadelphia. She was a weaver, her occupation necessitating perfect sight, as she was required to weave sixty threads to the inch. The patient had always been healthy, and, previous to her present trouble, had a slight attack of grip, but this did not stop her from working. On a Sunday morning she woke up at three o'clock and vomited, and again at six. She had a little headache, but otherwise felt well. After a short nap in the afternoon she awoke to find herself completely blind, in which condition she remained for three months before she came under our observation.

Dr. Krall reported: O. D., cornea clear, pupil dilated (no drops) 7 mm., round; media clear, the nerve outline is distinct; atrophic cupping, white atrophy; vessels small; macular region shows disturbance of pigment. V = 10 cm. 60.

O. S., V = 10 cm. 50. General appearance similar to O. D., except a small regular round patch of choroiditis in the inferior and temporal side. The reactions to light, accommodation and convergence were sluggish in both eyes.

When examined by me, except for the loss of vision, she did not complain of headache, nausea, or vomiting. She was however, irritable, would become easily excited, and complained of feeling "weak" all over the body. Station and gait with eyes open or shut were normal, but she had a tendency to stagger a little with her eyes shut. She could wrinkle her brows evenly on both sides, but on showing her teeth, either on one side or bilaterally, fibrillary tremors were noticed in the facial distribution, with some weakness. There was no other involvement of the cranial nerves. Hearing was acute. Taste and smell were not disturbed. Power was diminished in both upper and lower limbs, a little more on the left. The tendon reflexes were prompter than normal, a little more so on the right side. Sensation was normal for all forms. Some ataxia was present in both upper limbs, and about equally so.

Associated ocular movements were well performed downward and to the right, but associated movements to the left and upward were limited, especially in looking upward, where every attempt would be accompanied by a wrinkling of the brow. No hysterical stigmata were found at this time.

Because of the paresis of associated ocular movement to the left and upward, the apparent weakness of either seventh nerve and the optic atrophy, a tumor near the oculomotor nucleus was thought of. The symptoms were, however, by no means clear, and the diagnosis of hysteria was held in reserve.

A few days afterward examination denoted entire absence of paralysis of associated ocular movement either laterally or upward, and a further examination by Dr. Krall showed that vision could be improved considerably by suggestion. Although the patient stated that she could not see, objects purposely placed in her way would be properly avoided, and she would occasionally recognize fine type, while at other times this could not be recognized.

The apparent atrophic cupping of the optic disks may have been physiological, although their appearance was typical of such degeneration. The patient improved considerably under treatment, but passed out of our care, so it is impossible for us to say how far she finally improved.

Case II.—A single girl of twenty years, whose family history was negative. The patient had always been nervous and easily offended, but had otherwise been well, except for occipital headaches, which she had two or three times weekly since her twelfth year. A year before she came under observation she had her appendix removed, since which time she has complained of severe headache, nausea, vomiting, and vertigo. These symptoms became exaggerated during the six weeks before her admission to the University Hospital, and she also then first showed some inco-ordination in walking, which later developed into complete inability to walk, and she was confined to her bed. She also complained at this time of diplopia, and her physicians naturally thought of brain tumor. Dr. Spiller was called in consultation by Dr. Applebach, and she was admitted to the University Hospital.

At this time the patient complained of headache of a sharp character, which she described as resembling the noise made by the passing of a train of cars. Nausea, vertigo, and vomiting were also complained of. She was unable to stand without support, and would stagger on attempting to walk, but it was doubtful whether she would have fallen. During the examination the patient frequently jerked all over the body, and would throw herself around the bed. The eye examination showed a hippus on either side. The visual fields were normal. The patient later complained of temporary right monocular diplopia. The optic disks, except for a slight congestion of the vessels, were normal. The cranial nerves were normal. The limbs were a little weak, but resistance to passive movements could be increased on encouragement. The tendon reflexes of both upper and lower limbs were exaggerated. There was no ankle clonus or Babinski response. There was no atrophy anywhere. Sensation for touch was normal all over the body, but pain sensation was felt more acutely on the left side. Later touch sensation was more acute on the right and pain on the left. Hearing was acute. The sense of smell was lost and taste impaired. The right nostril was more sensitive than the left. The pharyngeal and conjunctival reflexes were preserved. She had marked inguinal and inframammary tenderness on the left, but not on the right.

Because of the manifest exaggeration of the symptoms as in walking, the presence of such hysterical stigmata as inframammary and inguinal tenderness, loss of sense of smell and impairment of taste, disturbance of sensation and the absence of organic symptoms, the diagnosis of hysteria was made. Suggestive treatment soon effected a cure.

Later the patient admitted that she had for the past year frequently discussed the symptomatology of brain tumor with a girl friend who did have such a neoplasm, and that gradually she began to complain of the same symptoms. It was not until an *x*-ray picture conclusively proved to her that such was not the case that she accepted the fact that she had no brain tumor.

These two cases illustrate well the difficulties encountered in diagnosticating brain tumor. At the first examination in each brain tumor was thought of, although a suspicion was aroused because of the bearing of the patients and the rather unusual symptomatology, as in the first case. The second case had the typical symptoms of brain tumor, and only careful observation permitted a correct diagnosis.

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MULTIPLE MOTOR NEURITIS, INCLUDING LANDRY'S PARALYSIS AND LEAD PALSY;*

WITH REPORTS OF CASES.

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CASES of rapidly developing paralysis of all four limbs are not common. Undoubtedly many classed as Landry's paralysis are caused by neuritis, but to assume that all have this etiology, as some writers have done, is unjustifiable. The literature of Landry's paralysis has become so extensive that it is inadvisable to review it. The subject has recently been discussed by Hans Lohrisch,[1] and the report of his case, among others, shows that Landry's paralysis may be spinal in origin. Lohrisch found disease of the gray matter in all parts of the spinal cord and medulla oblongata, as shown by hemorrhages, degeneration of medullary sheaths in the gray matter, degeneration of the nerve cells and of the anterior roots. He describes it as a diffuse acute poliomyelitis. The peripheral nerves were not examined. The patient had tabes also. There seems to have been no round-cell infiltration in the spinal cord.

The case of acute, rapidly-developing paralysis like that of Landry's paralysis, reported by one of

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us (Dr. Spiller[2]) in association with Dr. Sherman, showed much more intense alteration of the spinal cord than that reported by Lohrisch.

A recent case of acute ascending paralysis, with necropsy, reported by Edwin Bramwell,[3] is explained by him as the result of a toxic process acting upon the lower neurones. Degenerative changes were found in the facial and external popliteal nerves and tibialis anticus muscle. Bilateral facial palsy was an unusual complication of the acute palsy, and the paralysis first appeared in the face. The histological examination of the tissues for bacteria was negative.

Landry's paralysis is not always fatal. Some years ago one of us (Dr. Spiller[4]) reported a case of this type in which complete recovery occurred, and similar cases are recorded in the literature.

Buzzard[5] says that enlargement of the spleen and extensive pleurisy in the form of old or recent adhesions are found frequently in Landry's paralysis, and these lesions seem to indicate an infection.

A case of acute anterior poliomyelitis causing rapidly ascending paralysis has been reported recently by F. A. Hoch[6] with a review of the literature of this disease.

A case by Mills and Spiller[7] in which the peripheral motor neurones alone were affected was reported, with a careful review of the literature of the subject, in 1898. A still more recent paper on Landry's paralysis is by Peter Bassoe.[8]

Instead of discussing the subject of Landry's paralysis separately, we have preferred to include it under the heading of multiple motor neuritis. As already said, we do not ignore the spinal form of this symptom-complex.

The selective action of poisons for certain portions of the central or peripheral nervous system is one of the most striking facts of medicine. In uremia, whatever the intoxication may be, we see at times focal cerebral symptoms, and we are in the habit of explaining these on the theory of a locus minoris resistentiæ. Unquestionably similar symptoms are caused by arteriosclerosis, and we have been familiar with these at least more than ten years and have frequently referred to them; but to explain all uremic focal symptoms as the result of arteriosclerosis is unwarrantable.

In tabes, a metasyphilitic disease, we have the action of some poison on the posterior roots. Why the posterior roots should be implicated instead of the anterior we cannot say.

In tetanus we have the implication of the motor system, and in some cases paralysis of the facial nerve, a motor nerve. Alcohol and nicotine select the papillomacular bundle; diphtheria especially the nerve fibers to the ciliary muscle, and lead the distribution of the posterior interosseous nerve. Cramer[9] has referred to the fact that Arneth, on account of the appearance of the neutrophile leucocytes in the agonal period of Landry's paralysis and tetanus, made the inference that in both affections the central nervous system forms the point of attack of the infection. As Arneth puts it: "We shall hardly err if we ascribe to the poison of Landry's paralysis an exclusive affinity for the central nervous system."

Peculiar to Landry's paralysis is its irresistible course. Von Leyden in 1893 said that multiple neuritis requires less time for its development, and may become manifest very rapidly, and shows little tendency to the orderly extension, the gradual and steady progression, of Landry's palsy. Still, multiple neuritis of the ordinary type is at times confined to the lower limbs, in which Landry's paralysis usually develops, as in a recent case, with necropsy, observed by one of us.

Believing, as we do, that many cases with the symptom-complex known as Landry's paralysis are cases of multiple motor neuritis, we must seek a cause for the first manifestations of the paralysis in the lower limbs. The ingenious theory of Edinger may give us the answer. The nerves of the lower limbs are most employed, and therefore are most susceptible to poisons, and we have in Landry's paralysis, as in tabes resulting from another poison, the symptoms first manifested in the lower limbs.

We have recently had an opportunity to observe three cases that might be classed as Landry's paralysis, two with necropsy, but we prefer to regard them as atypical forms of multiple motor neuritis, atypical in the rapidity of their development. Multiple neuritis sometimes develops very rapidly, and one of us (Dr. Spiller) has had recently in his service at the Philadelphia General Hospital a case in which complete paralysis of all four limbs with great tenderness of all the muscles of the limbs to pressure, developed in about twenty-four hours.

These three cases resemble one another closely in the rapidly developing motor paralysis. The sensory symptoms in the first two cases, in the opinion of some, would prevent the diagnosis of Landry's paralysis, but those sensory symptoms were much less pronounced than the motor. We have described the three cases together to show the common pathological basis. Sensory symptoms were not excluded by Landry. There is much confusion regarding what should be called Landry's paralysis, as shown by E. W. Taylor and J. E. Clark.[10]

In the first case (a man 52 years of age, a laborer, of good habits) the condition started with numbness in the right hand and slight disability. Twelve hours later the hands were paralyzed, the legs weak, while in thirty-six hours a complete paralysis of all four limbs had developed. The paralysis was accompanied by pain in the calves of the legs and in the shoulders. These pains persisted ten days. The patient was examined four weeks after the onset of the disease; at this time there was complete flaccid paralysis of both upper and lower extremities, with absence of deep reflexes. The muscles of the arms and calves and the ulnar nerve were slightly tender to pressure. The sensations of temperature, pressure, and pain were normal. Control of the bladder and rectum was preserved. The head was not involved; the eyegrounds were normal; there was no fever. The muscles showed no marked alterations to electrical stimuli. After several months a small amount of voluntary motion of all four limbs was regained.

In the second and third cases death occurred and the course of the disease was rapid.

The second patient was a man, 60 years old, who had been a moderate drinker; he developed a sudden paralysis of both arms and legs. There were no premonitory symptoms, the patient waking one morning to find himself helpless. This paralysis was accompanied by numbness, pain in the lumbar region which shot down the inner side of the thigh to the toes, and pain in the arms. There was dyspnea, inability to cough, and difficulty in starting urination. On admission to the hospital two days later there was complete paralysis of the upper and lower extremities and of the diaphragm. The deep reflexes were absent. Sensation was unimpaired in the upper extremities, but on the inside of the thighs areas of anesthesia were found to alternate with areas of hyperesthesia. The sensory symptoms were comparatively slight. The paralysis ascended rapidly. The shoulders became involved; the voice was lost; swallowing became impossible, until finally, four days after the onset, the patient suddenly had dyspnea and died. Just before death he had incontinence of urine and feces. There was no fever. The autopsy findings as regards the nervous system were entirely negative. There was chronic aortic endocarditis and chronic nephritis. Macroscopic and microscopic examination of the spinal cord and brain showed no lesions. The peripheral nerves were not obtained. Bacteriological examination proved negative.

The third case differs from the other two in that there were no sensory disturbances, which, however, were not intense in the other two cases. The patient, a man 60 years old, who had been a painter for fourteen years but had not had lead colic, felt one evening a heavy sensation in his legs. The next morning his legs were weak, and in twenty-four hours they were partially paralyzed. The paralysis rapidly ascended. In two days the arms were affected and there was difficulty in swallowing. There was no pain and no incontinence of urine or feces. He was admitted to the hospital five days after the onset, and showed then almost complete paralysis of both upper and lower extremities with loss of all the deep reflexes except for a slight plantar response. Sensations were perfect. The paralysis rapidly became more profound and ascending and involved the muscles of the thorax and shoulders. The voice was lost, swallowing became impossible, dyspnea developed, and on the eighth day after the onset there was sudden cyanosis and death. The temperature remained normal. At autopsy there was an acute bronchopneumonia and chronic nephritis. The brain could not be examined. The spinal cord appeared normal on macroscopic examination. A microscopic study showed slight but distinct degeneration of the internal plantar and one other nerve; extensive changes in the anterior horn cells of the lumbar region, and marked fatty degeneration of the gastrocnemius muscle. Again the bacteriological examination was negative.

Finally, in the last case, one of typical lead palsy, examination of the central and peripheral nervous systems showed extensive degeneration of the anterior horn cells in the lumbar and cervical regions of the spinal cord, with pronounced degenerative changes in the left brachial plexus and moderate degeneration of the ulnar nerve.

In the first case sensory symptoms were of short duration, whereas the motor symptoms were persistent; in the second case also the sensory symptoms were not very pronounced, and in the third case they were entirely absent throughout the course of the disease. The last of these three cases is one that belongs clearly to the group of Landry's paralysis, but the microscopical examination showed that the symptoms were caused by degeneration of the motor cells of the anterior horns of the spinal cord, of the motor fibers in the peripheral nerves, and of the muscles. This case is remarkable in that the recent degeneration of the muscles as shown by the Marchi method is intense and out of proportion to the alteration of the nerve cells of the anterior horns and to the degeneration of the nerves. To these three cases of motor neuritis, in all of which the paralysis of all four limbs developed within twenty-four or forty-eight hours, we have added the unpublished case of lead palsy, with necropsy, as an example of still another form of multiple motor neuritis.

To multiple neuritis also belong at least some of the cases of the neurotic form, or muscular atrophy.

The reports of these cases in detail are as follows:

Case I.—Gano, aged 52 years, was referred to one of us (Dr. Spiller) by Dr. D. W. Shelly of Ambler, Pa., and was admitted to the University Hospital February 27, 1905, when the following history was obtained: He has been a laborer all his life, working principally on a farm. He has been married twenty-two years; does not drink, smoke, or chew tobacco, and says he never has done so. He denies venereal diseases and has eight healthy children.

Four weeks previous to admission he first noticed numbness of his right hand about 6 p.m. He was unable to use his knife and fork properly at the supper table that evening, with his right hand, but had no difficulty with his left. About the time he was ready to retire he found that the numbness was becoming more marked, and he was unable to unbutton his clothes. The following morning he could not use either his left hand or his right, and numbness was felt in both hands and arms. The lower limbs also were first noticed to be weak and numb, and he had some difficulty in walking and was obliged to hold on to the furniture as well as he could with his weak hands. On the evening of the same day he attempted to go upstairs and found that he was unable to do so. His legs gave way beneath him and he was obliged to sleep downstairs. That evening he began to experience dull and sometimes shooting pains in his calves, in his shoulders, and back.

The following morning he was completely paralyzed in all his limbs, but had no difficulty in urination. The pains continued ten days, after which they disappeared.

He has not had incontinence of urine, but urination is a little slow, and the bowels are somewhat constipated. He had severe diarrhea three or four days before the paralysis developed.

The notes of the examination soon after his admission are as follows: He draws up his shoulders very slightly, and the right a little more than the left. There is very little distention of the abdomen when he breathes deeply, and he seems to have paralysis of the diaphragm. Respiration is very shallow and is mostly costal in type. When he attempts to breathe deeply both sternocleidomastoid muscles contract. The upper limbs are completely paralyzed and flaccid. The skin of the hands is rough and desquamative, especially on the palmar surfaces. The biceps and triceps tendon reflexes are completely lost on each side. The sensations of touch, pain, and temperature are normal in both upper limbs and over the front of the trunk. The muscles of the upper limbs and the ulnar nerves are tender to pressure. Squeezing the hands causes much pain.

The lower limbs are completely paralyzed except that the man can draw each limb upward a little at the hip. The patellar tendon and Achilles tendon reflexes are lost on each side. Babinski's reflex is not obtained, the toes not moving in either direction from irritation of the sole of the foot. Both calves and feet are very tender to pressure. Sensations for touch, pain, and temperature are normal in the lower limbs and there is no distinct atrophy of these limbs.

March 6, 1905. The cranial nerves are not implicated, and sensation in the face is normal. The pupils are equal, and the pupillary responses are normal. The head is moved freely from side to side. The heart, lungs, and abdominal viscera seem to be normal.

Dr. de Schweinitz's examination of the eyes revealed nothing abnormal. Dr. Potts made an electrical examination and reported as follows:

"March 16, 1905. *Right leg:* All muscles respond to a strong faradic current (all but one inch of secondary coil) excepting peroneals and rectus femoris, which do not respond. *Flexor longus digitorum* requires somewhat weaker current than the other muscles.

"*Left leg:* All respond to strong faradic current, slightly better than those of right leg.

"*Right arm:* All muscles, also those of shoulder, pectorals, and abdominal muscles respond to strong faradic current, but not so strong a current is required as for the leg (secondary coil two inches from end).

"*Left arm:* All muscles respond to slightly weaker current than is required for the right arm.

"*Right leg:* Tibialis anticus, flexor longus digitorum, gastrocnemius, soleus, show quantitative decrease. K Cl C > An Cl C. No modal change. Peroneals quantitative decrease. An Cl C > K Cl C. Modal change. Rectus femoris, An Cl C > K Cl C. Modal change. Vastus internus, An Cl C > K Cl C. Other muscles of thigh, K Cl C > An Cl C. Tendency to modal change in adductors, irritability about normal.

"*Left leg:* In muscles below the knee, K Cl C > An Cl C. In soleus slight modal change, quantitative decrease. In vastus externus, An Cl C > K Cl C. Slight modal change in other muscles of thigh, no change in irritability.

"*Right arm:* K Cl C > An Cl C in all muscles, irritability about normal, excepting in ulnar distribution, where it is increased and An Cl C is relatively greater than normal.

"The same holds good for the left arm.

"With the faradic current the muscular contractions did not cause any motor effect. Galvanic current did cause motor effect in arms, but not in legs."

The patient did not have any fever, and after some months regained a small amount of voluntary movement in all the limbs, sensory disturbances having entirely disappeared.

Case II.—Dr. C. C., 60 years of age, physician, was a patient in the service of Dr. A. V. Meigs at the Pennsylvania Hospital. On admission he complained of paralysis of the arms and legs.

Family History: Father died of apoplexy, aged 84; mother died of old age at 88; one brother living, aged 63, has hemiplegia; one sister died recently from dropsy and hemorrhage from the bowel. Three daughters and one son living and well. One uncle died of apoplexy; one brother living and well, a minister.

Personal History: Had typhoid fever when 16 years of age; has had no other disease. Was graduated from the University of Pennsylvania Medical School in 1870, then practised in Virginia, later in Iowa, and for the last fifteen years in Philadelphia. Uses alcohol moderately; chews tobacco moderately; no drugs (?). Had lumbago, which stiffened his legs, about ten years ago. No venereal history.

Present Illness: About two years ago the patient awoke in the morning with severe "lumbago," which also passed around his waist and made his legs stiff so that he could not get out of bed. He also had pain in his hips and numbness in his feet. He was in bed with the attack four or five days and gradually improved. Since then he has had no trouble with his legs at all. Day before yesterday when he awoke in the morning he found that he could not move his arms or legs; he had been perfectly well the evening before, and can think of nothing he had done in the direction of unusual exertion, except a fall on his hip five days before. He has had since the onset a feeling as of pins and needles in his extremities, and pain in the lumbar region, which shoots down the inside of his thighs to his toes when he is moved. This morning he has had slight pain running down the inside of his arms. He has had no headache, loss of consciousness or convulsions. Bowels have not moved since the attack; has had slight difficulty in starting urination. There is dyspnea, inability to cough, but no dysphagia. He has had no trouble with vision. (Had a blow in his right eye in 1889 and three years later Dr. Harlan did a tenotomy; the eye has been blind since it was struck.)

Physical Examination: Patient is a large, well-nourished man, lying on his back; slightly dyspneic, apparently employing only the upper part of his chest; abdomen retracts with each inspiration (no Lytton's sign); skin of trunk and extremities covered with an eruption consisting of small crusts, evidently arising from scratching. Urine: dark amber; mucoid sediment; acid, 1,028; no albumin or sugar; few hyaline casts. On the shins are three or four large pustular lesions covered by dark crusts. Right eye: (optic atrophy) has been blind for eighteen years; pupil small and immobile, internal strabismus. Left eye: pupil is eccentric, being nearer the lower part of the cornea; apparently it reacts normally. No facial paralysis; tongue slightly coated, protruded straight, no tremors. Movements of neck normal except that he cannot raise his head very high. He can flex the fingers slightly and extend the right leg slightly. He can also raise his shoulder. All tendon reflexes in the extremities are absent. No plantar reflex; no Babinski; tactile and pain sensations in the upper extremities are normal. There are irregular areas of anesthesia in the thighs, and in other areas there is apparently hyperesthesia. The patient complains of a great deal of pain in his hips. Lungs: anteriorly are normal. Heart: O. C. D. normal, sounds clear, regular and of fair tone. Liver and spleen: negative. Abdomen: negative.

January 22. Patient died yesterday about 4 P.M. About 2:30 P.M. he suddenly became exceedingly dyspneic and cyanosed. The pulse remained very strong and he was conscious until late. In the morning he was unable to raise his shoulders or to speak above a whisper, and complained of heaviness of his chest. At noon he was unable to swallow; had incontinence of urine; bowels did not move. Average temperature, 97; average respiration, 25; average pulse, 90.

Autopsy No. 652; Hospital No. 3,311. January 22, 1905. *Anatomical Diagnosis:* Chronic diffuse nephritis; chronic aortic endocarditis; cardiac hypertrophy; acute splenic tumor; cloudy swelling of liver; congestion of brain; congestion of viscera; rupia (?).

The body is that of a fairly well-built old man, 172 cm. in length. Rigor mortis well marked. Much purplish discoloration of dependent parts. Left pupil dilated, right contracted. On arms, chest, back, abdomen, and legs are numerous small, raised black scabs, averaging about the size of a pinhead and occasionally running into streaks. On the inner surface of the right arm, most marked at the elbow, are very small ecchymotic spots. Over the anterior surface of both tibiæ there are several large raised crusts, varying from 1 to 2.5 cm. in diameter. Six of these are on the right and one on the left leg, from which the scabs have been removed. The scabs are piled up towards the center, having a slightly purplish skin margin, which is dried and over which the epidermis scales off in concentric rings. External genitalia apparently normal. The abdominal fat measures 3 cm. in thickness and is orange colored. The muscles are rather pale.

Abdominal Cavity: The omentum is small and contains a good deal of fat. Peritoneal surfaces are smooth and glistening. The intestines are pinkish or gray in color and rather collapsed. Appendix measures 11 cm. in length, lies pointed upward behind the cecum in the retroperitoneal tissue, is patent to tip and apparently normal. The liver is bound to the diaphragm by fibrous adhesions. Diaphragm extends to third interspace on right side and to fourth interspace on the left side.

Thorax: The lungs are voluminous and meet in mid line. Both pleural cavities are free from fluid and adhesions. Pericardial cavity contains a small amount of clear, straw-colored fluid.

Heart: Weight, 390 grams. The heart is quite large, the cavities are filled with fluid blood and red post-mortem clots. The pericardium is smooth and glistening and contains a good deal of fat. The tricuspid and pulmonary valves are thin, delicate, and apparently normal. The mitral leaflets are also quite delicate; the aortic flaps, although large, are a little thickened and present calcified patches at the bases of the sinuses of Valsalva which stiffen the attachments of the leaflets. The heart muscle is firm, beefy, and red in color. The left ventricle is hypertrophied;

the wall measures from 15 to 20 mm. in thickness, and the cavity is small. The aorta is large; the wall is not thickened, and the surface is exceedingly smooth. Coronary arteries are large and patulous. Auricular appendages are free from thrombi.

Lungs: The left lung is fairly large, rather heavy and soggy, especially in the posterior portion. The anterior portion of the upper lobe is lobulated, mottled dark gray, red, and black. Posterior portion of the upper lobe and lower lobe present a smooth, bluish-gray surface. The pleura is everywhere smooth and glistening. Lung is everywhere crepitant. On section cut surface oozes blood and frothy fluid, is deep purplish-red in color, soft and crepitant, except for a few indefinite red patches in the lower lobe. Bronchi at root are congested and contain bloody mucus. Vessels are clear. The right lung is in general like the left. The lower lobe is quite heavy and soggy. Crepitation, though present, is generally decreased. On section there is general congestion and edema, and one sees indefinite small solid patches in the lower lobe and a few more definite, raised, grayish, granular patches in the upper lobe. Bronchi and vessels at root are like right lung.

Spleen: Weight, 120 grams; measures 13 x 12 x 4 cm. The spleen is of medium size, regular, exceedingly soft and mushy. Color is a pale violet. The capsule is roughened and thickened in many places. Posteriorly it is firmly adherent to the diaphragm. On section the pulp is soft, almost diffluent and of a pale, muddy, red color. Malpighian bodies seem to be very large and indefinite; trabeculæ not very prominent.

Liver: Weight, 1,680 grams. Size, 26 x 18 x 10 cm. Liver is large, regular, and decreased in consistency. Capsule is adherent over a large portion of anterior surface to diaphragm and is torn in removing the organ. Color is a uniform light reddish-gray. On section the cut surface is smooth; the lobules are well marked, the centers being red and the peripheries gray, so that the surface has a slightly fine mottled appearance and is generally opaque and cloudy. Portal connective tissue is not specially increased. Bile ducts are patent. Gall-bladder is apparently normal.

Kidneys: Combined weight, 220 grams. The left measures 12 x 4.5 x 5 cm. It is fairly large, regular, and firm. The capsule strips with a little difficulty, leaving a finely granular, dark red surface showing a few injected stellate veins. Here and there, three small yellow points the size of a pinhead are seen. On section the kidney substance is decreased in proportion to the pelvic fat, but is still present in fair amount. The striæ are well marked, usually irregular, occasionally a little distorted. The medullary pyramids are large, dark, and streaked with purple. The cortex is from 6 to 10 mm. in thickness and is red in color. Vessels are injected. The glomeruli are large and purple in color. Pelvis and ureter apparently normal. The right kidney measures 12 x 6 x 3.5 cm. The surface shows two small yellow pits. In all other respects the kidney is like the left. Adrenals are apparently normal. Testes apparently normal. Urinary bladder, prostate, seminal vesicles apparently normal.

Stomach and Intestines appear normal except for some congestion.

Brain: The brain is of medium size and fairly regular, although bulging a little over the parietal lobes. The dura mater is thickened and adherent to the pia in places. The pia is slightly thickened. The vessels are congested. At the base the vessels are very delicate and collapsible. The ventricles are not dilated. No macroscopic lesions are found.

Spinal Cord: Both the dura and pia mater are delicate; there are no adhesions between them. Vessels are congested. The general appearance is normal. On section the white matter seems very faintly mottled in many places by yellowish patches. This is irregular, but seems to affect most of the posterior portion of the cord. Left eye appears normal.

Aorta: The ascendnig arch is quite smooth; in the descending arch, about the opening of the greater vessels, there is an irregular, circumscribed patch about 2 cm. in diameter, which is yellow or cream-colored, raised, scarred, softened, and in some places calcified. Throughout the abdominal aorta there are localized, nodular, yellowish and cream-colored swellings varying from the size of a pea to 1 cm. in diameter; they are rather soft and have a fairly smooth surface. They are particularly numerous about the openings of the blood-vessels. Iliac vessels are free from thrombi. There is no general enlargement of the lymph nodes.

Microscopical Examination.—Heart: There is an excess of pigment about the nuclei of muscle fibers, with some fatty degeneration; no increase in connective tissue.

Lungs show marked congestion, and in places the alveoli contain large cells filled with brownish-black pigment, a little coagulated serum, red blood corpuscles, and polymorphonuclear leucocytes.

Spleen: The capsule is thickened and contains a good deal of yellowish-brown pigment. The pulp shows a patchy congestion, with general hyperplasia of the cells. The Malpighian bodies are large and in some the germinal centers are swollen and show central necrosis. There is some thickening of the intima of the blood-vessels.

Liver: There is well-marked congestion, with some parenchymatous and fatty degeneration of the liver cells. The portal connective tissue is slightly increased.

Kidney: There is much congestion, and the cortex presents slight proliferative changes. In the connective tissue descending from the capsule there are many small round cells. The tubular epithelium shows parenchymatous degeneration. Most of the glomeruli are engorged with blood; a few have undergone complete fibroid change.

Aorta: There is an extreme grade of irregular fibrous thickening of the intima, with fatty degeneration, deposits of cholesterin crystals and calcified material. The media suffers little change, though there is some degeneration of the elastic fibers.

Sections of the spinal cord, stained in hematoxylin and acid fuchsin (midlumbar and cervical regions). No definite alterations can be made out. The vessels of the pia and the smaller vessels of the cord are delicate and show no thickening. The cells of the anterior horns stain well by the thionin method, but are much pigmented. Marchi sections also are normal. Unfortunately peripheral nerves were not obtained.

Case III.—A. L., 60 years of age, painter; was in the service of Dr. A. V. Meigs, Pennsylvania Hospital. Complains of paralysis of arms and legs.

Family History: Father died of diabetes; mother of accident; two sisters have diabetes. No other family disease; no nervous disease. Has three healthy children.

Personal History: Has never been sick in his life, except for a few slight attacks of rheumatism. Had gonorrhea when about fifteen years old; never had chancre; drinks whiskey occasionally, but never to excess; never had lead colic; no injury in recent years (barrel fell on him many years ago). He has been a painter for fourteen years and at the onset of this attack was in perfect health.

Present Illness: Last Friday night (five days ago) patient noticed, while going upstairs, that he had a feeling as if heavy weights were in his hip pocket; also when he stooped down he had difficulty in rising. He slept well that night, and had no discomfort, but in the morning found that his legs were so weak that he could not stand to dress himself and he could put on only one of his shoes. By evening he could not raise himself because of weakness in his back. On Sunday his hands and arms began to be weak, and swallowing became difficult. Since then there has been no change except that the existing paralysis has become more complete. There has been absolutely no pain since the beginning of the attack. There has been no loss of sensation, or perverted sensation; no loss of control of the bladder or rectum. No dyspnea. Bowels have been moved by laxatives.

Urine: amber color, clear, acid, 1035; albumin, very faint trace; no sugar, few hyaline casts. Hemoglobin, 95. Leucocytes, 4,900.

January 24.—*Physical Examination.*—Patient is a fairly well-nourished old man, apparently no older than he is; speaks distinctly and intelligently; soon becomes tired of lying in any one position, but otherwise has no complaint. Pupils and movements of eyeballs are normal; no facial paralysis; can turn his head from side to side, but cannot raise it from the pillow. Tongue is protruded straight; some difficulty in swallowing, but he is able to get down liquids fairly well; is able to shrug his shoulders and can move his right arm a little, but grip with the right hand is very weak. Complete paralysis of the left arm; sensation as elsewhere apparently normal for tactile and pain sensations. Abdomen protrudes with each inspiration (Lytton's sign present); can extend the toes and move both ankles very slightly; plantar reflex present to a slight degree; no Babinski; both knee jerks absent; no ankle clonus; no apparent atrophy; control of bladder and rectum normal.

January 25. Had a restless night; swallowing difficult; paralysis about the same; voice not so strong as yesterday; no pain; much mucus in throat.

January 26. Sleeps very little, mind perfectly clear; voice weaker, sometimes whispering; grip of right hand is noticeably weaker; is fed from a medicine dropper; had to be catheterized for the first time this evening; no difficulty in respiration, but complains very much of the mucus which collects in his throat.

January 27. Cannot speak above a whisper today; had to be catheterized this morning, but later urinated involuntarily. Bowels have not moved since admission; cannot move his right hand this evening; about 4 P.M. became cyanosed and complained of dyspnea; also of pain in his legs, and the pain later moved to the small of his back. Entirely unable to swallow since noon; chest does not expand with respiration; diaphragm moves; slightly irrational this P.M.; pulse weak and rapid; profuse perspiration.

January 28. Died at 10:10 P.M. For three hours previously had been semi-stuporous, but was able to answer questions; had to be propped up to get his breath; pulse very weak and rapid; breathing stertorous towards the end, but was regular; this suddenly became shallow and in less than a minute ceased entirely.

Average temperature, 99; respiration, 28; pulse, 90.

Autopsy. No. 653. Hospital No. 3378.

A. L. January 28, 1905; aged 60 years; performed by Dr. Longcope.

Anatomical Diagnosis. — Acute bronchopneumonia; congestion of liver, kidneys, and abdominal viscera; chronic interstital splenitis and hepatitis; chronic diffuse nephritis; chronic fibrous pleurisy.

The body is that of a well-developed man, 170 cm. in length. Rigor mortis is marked. Slight postmortem discoloration over dependent parts. Pupils equal and dilated. External genitalia normal. No edema of extremities.

Abdominal Cavity: The omentum is quite large, contains a good deal of fat, and lies in the upper part of the cavity. Peritoneal surfaces are smooth and glistening, but slightly injected, mottled pink, and brownish yellow. The appendix measures 5 cm. in length hangs over the brim of the pelvis, is free, and the lumen is obliterated throughout. Diaphragm extends to the fifth space on the right side and to the fifth rib on the left side.

Thorax: Lungs are voluminous and collapse on removal of the sternum. The right pleural cavity is free from fluid and adhesions. The left pleural cavity contains cobweb-like fibrous adhesions. Pericardial cavity contains a slight excess of clear, straw-colored fluid; serous surfaces are smooth and glistening.

Heart: Weight, 240 grams. The heart is of medium size, epicardium is smooth and glistening, contains a good deal of fat, and shows at one point over the right ventricle a milk patch the size of a quarter. The vessels are prominent and the lymphatics somewhat dilated. Cavities are filled with soft, red post-mortem clots, but are not greatly dilated. All the valves are exceedingly thin, delicate, and normal. The endocardium is everywhere thickened and gray in color. The heart muscle is fairly firm, cloudy, and brownish-red in color. The left ventricle averages from 12 to 15 mm. in thickness. Coronary arteries are patulous and their walls are fairly delicate. The aorta is quite smooth and delicate except for a few yellowish cross-streaks above the aortic leaflets. Auricular appendages are free from thrombi.

Lungs: The left lung is large, rather heavy and soggy. Anteriorly the color is gray and the lung is lobulated. Posteriorly it is smooth and deep purplish in color. Over the apex the pleura shows small, thick, white cartilaginous areas of thickening, and in one or two other points there are similar patches two or three centimeters in diameter. There is some air everywhere throughout the lung. On section the cut surface is deep purplish-red in color and oozes frothy blood and serum. Scattered everywhere throughout the lower lobe are numerous small raised purplish-gray areas, slightly granular and firm; they average about 0.5 cm. in diameter. Bronchi at the root contain some bloody mucus. Vessels are clear. The right lung is rather small, somewhat retracted, and the pleura is covered with fresh fibrous adhesions, so that the lung is torn in many places during removal. The organ is heavy and contains very little air, the lower lobe being almost entirely solid, but at the same time rather soft and flabby. On section the lung presents a deep purplish-red, moist surface, from all parts of which bloody fluid filled with air bubbles exudes. The middle lobe and anterior portions of the upper lobe are crepitant. The remainder of the lung presents large, irregular, solid patches which are sometimes smooth and red, but more often raised, granular, and grayish. Between these areas of consolidation there is usually a little air contained in the lung. Bronchi at the root are congested and contain a little mucus. Vessels are clear.

Spleen: Weight, 90 grams; size, 10 x 7.5 x 4 cm. The spleen is small, regular, and much decreased in consistency. The color is pale lilac. Capsule is wrinkled, shows a little thickening, and is free from fibrous tags. On section the cut surface is a little rough, trabeculæ being coarse, increased, and prominent. The color is mottled gray and red. Malpighian bodies are quite large and pale.

Liver: Weight, 1,410 grams; size, 24 x 21 x 6.5 cm. The liver is of medium size, fairly regular, and quite firm. Over the anterior surface of the right lobe the capsule shows a scar-like area of thickening, about 8 cm. in diameter; this does not extend into the liver substance. There is also a little thickening over the anterior surface of the left lobe; elsewhere the capsule is delicate, although the surface is a little irregular. The color is a uniform brown. On section the cut surface is slightly granular, brown mottled with red. The lobules are well marked and regular, their centers are red, peripheries brown. Portal connective tissue is a little increased. Bile ducts patent. Gall-bladder is normal.

Kidneys: Combined weight, 250 grams. The left measures 12 x 5 x 4 cm. It is of medium size, regular and firm. The capsule is slightly thickened, strips with difficulty, leaving a finely granular, uniform purplish-red surface. On section the kidney substance is slightly decreased in proportion to pelvic fat. The medullary pyramids are fairly large, slightly irregular, and streaked with purple; cortex is deep red and well defined, is somewhat irregular and measures from 4 to 8 mm. in thickness. The striæ are well marked and regular; glomeruli are injected. Pelvis and ureter apparently normal. The right kidney is like the left. Adrenals are apparently normal. The urinary bladder contains several cubic centimeters of cloudy urine; mucous membrane is deeply congested and purplish-black in color about the trigonum. The prostate is large across the lateral lobes, which measure 5 cm. from right to left. On section it is soft and has a spongy appearance. Seminal vesicles and testes apparently normal. Pancreas is apparently normal.

Stomach is congested and shows hemorrhages on the crests of the rugæ.

Intestines are apparently normal. The *aorta* is quite smooth throughout the arch. In the thoracic portion just above the bifurcation of the iliacs, the wall is partly calcified, but thin, and the intimal surface is smooth. Iliac vessels are free from thrombi.

Spinal Cord shows some congestion of the pial vessels. The dura mater and pia are thin and delicate. There is a small amount of clear fluid in the arachnoid space, but otherwise normal to the naked eye. Muscles of the calf appear normal. Permission to open the skull could not be obtained.

Microscopical Examination. — Heart: Muscle fibers are small and show an excess of pigment about the nuclei. There is some increase in the connective tissue of the large trabeculæ.

Lungs show marked congestion and edema, with patches of bronchopneumonia where the alveoli are filled with leucocytes, red blood corpuscles, and a little fibrin.

Spleen shows a patchy congestion, with some increase in the connective tissue of the pulp. The Malpighian bodies are large and their margins ill-defined. There is some thickening of the intima of the blood-vessels.

Liver shows some congestion, with parenchymatous degeneration of the cells.

Kidney shows slight increase in connective tissue of the cortex, with swelling and parenchymatous degeneration of the cells of the convoluted tubules. The glomeruli are as a rule congested. Many hyaline casts are seen. The intima of the blood-vessels presents a moderate grade of thickening.

Bacteriological Examination. — Cultures made by

Dr. Robinson from the left lung yielded *Micrococcus lanceolatus* and an unidentified bacillus. Cultures from heart's blood showed no growth after forty-eight hours at 37° C.

Spinal Cord. Lumbar region: The sections appear to be normal except that in many of the nerve cells of the anterior horns the chromophilic elements have become granular and the nucleus is displaced to the periphery of the cell. The alteration of the nerve cells is distinct, but not intense. The pigmentation of the cells is pronounced. The anterior and posterior roots are normal. Cervical swelling: The condition of the spinal cord at this level is much the same as in the lumbar region. No alteration is seen by the Marchi method. Sections of the sciatic nerve are normal. Pieces of the right obturator, internal plantar, and posterior tibial nerves were examined, but the portions of nerves removed had become mixed. In two of these smaller nerves some degeneration is observed by the Weigert hematoxylin stain. Slight but distinct degeneration in the form of many black granules is found in the right internal plantar and the left internal popliteal nerves when fibers are teased in the fresh state and stained by osmic acid. Swollen axis cylinders are seen in one of the small nerves. When a nuclear stain is employed the muscle appears normal, except that there may be a slight increase in the sarcolemma nuclei. Nerve fibers within the muscle are normal.

Case IV.—J. L. A., 67 years of age, was at first in the service of Dr. Alfred Stengel, at the Philadelphia General Hospital, and was then transferred to the service of Dr. Spiller.

July 16, 1904. He had been a painter since he was 14 years of age. He first began to have weakness with his upper limbs in 1876, and has had six or seven attacks (probably of paralysis) in these limbs since. Two weeks ago he stopped work, complaining of diarrhea and loss of power in his upper limbs. He has not had headache nor pain anywhere. Has lost his appetite. He could walk when he had his shoes on without much difficulty. He is emaciated and anemic, and his face appears edematous. Pupils are equal and react sluggishly. He has no blue line on the gums. Pulses are equal and easily compressible; vessels do not show much sclerosis. Chest is somewhat emaciated, supraclavicular spaces are sunken, interspaces are wider than normal. Lungs are resonant and no râles are detected. Rough breathing is heard over the anterior surface of the right lung at the upper part. Heart dullness is about normal in outline; sounds are not very strong; no murmurs. Liver dullness extends from the upper border of the sixth rib to two fingers' breadths below the costal border. Spleen not palpable. Epigastric pulsation is marked.

He has bilateral wrist drop, with much atrophy of thenar and hypothenar eminences. In the lower extremities there is palsy and atrophy of the peroneal groups of muscles. The skin of the feet and legs is glossy, and there is some swelling and pitting upon pressure. He shows a marked intention tremor of the hands. Patellar tendon reflex is diminished; biceps tendon reflex seems to be normal. He has not had any pain or disturbance of vision.

August 15, 1904. He is very feeble, and examination is unsatisfactory because of stupor. He does not obey commands. He moves his head freely from side to side; does not take his nourishment now, but has not had any difficulty in swallowing. The wrist drop is very pronounced on each side, but he is not paralyzed in the muscles of the shoulders, as he can voluntarily place both upper limbs above his head. The fingers of each hand, especially those of the right hand, are somewhat cyanotic. The biceps tendon reflex is weak on the left side and uncertain on the right. The triceps tendon reflex is not obtained on either side. The muscles of the arm of each side are wasted, but not so much as those of the forearms. Each forearm and each hand are much wasted, but the extensors on the back of the forearms are more wasted than the flexors. He has not been seen to move his lower limbs, although he has some power in the muscles of each thigh. The lower limbs are flaccid except at the ankles. The feet are in the position of talipes equino-varus and cannot be passively straightened. The lower limbs are much wasted, especially in the peroneal group of muscles. The patellar reflex is entirely lost on each side. Achilles tendon reflexes cannot be obtained. Babinski's reflex is uncertain on the right side, there being no distinct movement of the toes on account of the contractures. This reflex cannot be tested on the left side, because of an amputation at the metatarsophalangeal articulations. He has incontinence of urine and feces. His mentality is much obtunded.

Pathological Diagnosis.—Brown atrophy of heart, senile emphysema, chronic interstitial nephritis.

Microscopical Examination. — Lumbar Region: The nerve cells of the anterior horns are much altered; the chromophilic elements in most of the cells, instead of being rod-shaped, appear as fine granules. The posterior and anterior roots are normal. There are no signs of meningitis. No degeneration can be detected by the Marchi method.

Midthoracic Region: Sections from this region present no secondary degeneration of the posterior columns, and the posterior roots at this level are normal.

Cervical Region: Sections stained by thionin show disintegration of the chromophilic elements like that of the lumbar region. Outside of the changes in the nerve cells, sections of the cervical region seem to be normal.

Paracentral Lobule (left and right): The cells of Betz show slight disintegration of the chromophilic elements and much pigmentation, but on the whole the nerve cells are in a fair state of preservation. The proliferation of the endothelial cells covering the pia is not distinct in this case as it was in the former case of lead poisoning reported by one of us (Dr. Spiller), although a slight indication of it is detectable.

Left Brachial Plexus: Sections here show a pronounced partial degeneration both by the Weigert hematoxylin and acid fuchsin stains, but there is no degeneration by the Marchi method.

Pieces of the right ulnar nerve and of the left brachial plexus do not appear much degenerated by the 1 per cent. osmic acid stain. It is unfortunate that more peripheral nerves were not obtained.

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CARCINOMA OF THE NERVOUS SYSTEM, WITH THE REPORT OF ELEVEN CASES.*

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We have had the opportunity of studying eleven cases of carcinoma implicating the nervous system, eight of these were with necropsy. From a study of these cases and of others in the literature we have made the following classification:

First—Cases in which metastasis to the central nervous system occurs from a primary lesion elsewhere in the body.

Second—Those in which the alterations in the substance of the spinal cord and brain are not distinctly carcinomatous, but are such as may be caused by pressure, etc.

Third—Those in which the peripheral nerves are diseased either with or without involvement of the brain and cord.

Fourth—Those in which no macroscopic lesions are found, and possibly in some, no microscopic lesions, and the symptoms are supposedly toxic.

Metastasis in the central nervous system secondary to carcinoma elsewhere in the body may assume many forms. It may occur as a multiple carcinomatosis, which may or may not be a part of a general carcinomatous process elsewhere. There may be only a few cancer nodules within the central nervous system, or the process may involve the brain or cord and the meninges extensively, or the meninges alone. The meningeal infiltration may be either diffuse, as carcinomatous meningitis, like the sarcomatous meningitis, or may be in the form of multiple small tumors.

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Metastasis may occur anywhere in the brain. It is probable that cancer nodules occasionally are overlooked, either because of their resemblance to the surrounding brain tissue, or because of their minute size.

Metastasis to the substance of the spinal cord is extremely rare and is nearly always from the periphery of the cord. Metastasis in the vertebræ is more common.

In the second classification, in which alterations in the spinal cord or brain occur, which are not carcinomatous, the changes may be in the form of a diffuse sclerosis, or an acute inflammatory process. Occasionally in association with degenerative changes in the cord, there may be disease of the spinal roots and peripheral nerves.

In the third classification, viz., cases in which the peripheral nerves are diseased either with or without involvement of the brain and cord, the alterations in the peripheral nerves and muscles may be mild or severe. Raymond[1] has recently recorded a case in which the symptoms of multiple neuritis were present, but there was no cancerous invasion of the nerve trunks. The intramuscular nerve ramifications were diseased, and innumerable cancerous masses were found in the muscles; the condition was an acute miliary carcinomatosis, especially of the muscles.

In the fourth classification, in which the symptoms seem to be of toxic origin, may be included all those cases in which no gross pathological changes are found. The microscopic examination, however, may show alterations in the nerve cells in different parts of the brain and cord, sometimes a marked congestion of the blood vessels and degenerative changes in some of the cranial nerves. As toxic conditions, may be included the following: Hemiplegia, with or without aphasia, aphasia alone, monoplegia, convulsions, either general or Jacksonian in type; cranial nerve paralysis and bulbar phenomena. The psychoses which sometimes complicate carcinoma may also be placed here, although some authors believe that psychoses do not occur unless there be an intracranial metastasis.

It is noteworthy that in the majority of instances the symptoms are not so pronounced as might be expected from the pathological findings. This is either because many of the lesions produce no alteration of the surrounding tissue, or else the lesions, though apparently intense, are not sufficiently so to produce symptoms.

It will be impossible to consider in this paper the large amount of literature on the subject of carcinoma, and only the more important contributions will be referred to. It will be our purpose to dilate upon the classifications made, using such literature as is necessary and drawing our conclusions also from the cases we here record. It must of course be understood that the classification adopted is only relative, and that one case may have symptoms belonging to each division made.

Metastasis to the osseous and connective tissue coverings of the spinal cord occurs most often in sarcoma, and next in frequency in carcinoma. Schlesinger[2] in his monograph on tumors of the spinal cord and of the vertebræ, published in 1898, states that metastasis occurred to these coverings in 2.4% of the cases of carcinoma elsewhere in the body and in 8.8% of sarcoma elsewhere. His observations were drawn from 13,300 necropsies of all kinds. In another series of necropsy records of 35,000 cases carcinoma metastasis to the brain occurred as follows: cerebrum, 16; cerebellum, 8; medulla oblongata, 5; bones of the skull, 41; dura, 17; pia, 2; in all, 89 times. The conclusion can be drawn that metastasis of carcinoma to the nervous system occurs in the following order of frequency; vertebræ, bones of the skull, brain, and very rarely into the spinal cord.

In 1898 Buchholz[3] collected 66 cases of cancer of the brain, exclusive of tumors of the hypophysis and of the bones of the skull. Of these 41 were said to be secondary, 5 primary, and the remainder were more or less doubtful. Two of the primary growths originated in the epithelium of the fourth ventricle (Wunschheim[4]) or the aqueduct of Sylvius (Coats[5]). The three other cases are regarded by Buchholz as questionable. Besides these cases which are quoted by Buchholz, Ziegler is said to have recorded a papillomatous cancerous growth of the choroid plexus and Cornil and Ranvier a similar growth of the third ventricle. Another similar case of Spaet is often quoted in the literature, but we have been unable to obtain the original paper.

Metastasis occurs more often from a primary growth situated in the mammary gland, lung or pleura, in the order mentioned.

It also occurs secondary to cancer of the stomach, pancreas, ovaries, testicles, uterus and rectum.

Metastasis to the nervous system may occur:

1. Into the vertebræ.
2. Into the bones of the skull.
3. Into the substance of the brain, cord and meninges.
4. Into the brain alone.
5. Into the substance of the brain and cord.
6. Into the meninges alone.
7. Into the spinal cord alone.

Secondary carcinoma of the brain is often multiple, and may be part of a general carcinomatosis elsewhere, as in Case IV. of Gallavardin and Varay[6] in which from 150 to 250 cancer nodules were found in the brain, and others were in the cord. Metastasis may involve almost any portion of the brain, but the central convolutions seem to be frequently involved as in the cases of Blum[7] and Gallavardin and Varay[6] (Case I.).

Next in frequency are perhaps the cerebellar lobes as in the cases of Saenger[8] and Gallavardin and Varay[6] (Case II.). The temporal lobes are also sometimes involved, as in the case of Kufs[9] in which there was also a cancer of the pons and in Case II. of Gallavardin and Varay[6]. In Case I. of our series, a secondary growth of the hypophysis was found, as in the recent case of Götzel and Erdheim[10].

The cancer nodules may be as large as a hazel nut or even larger, or may be of microscopic size. When the nodules are small many may be overlooked unless a thorough microscopic examination is made. In multiple carcinomatosis of the nervous system, as in the cases of Buchholz[3], Siefert[11] and others, the cancer nodules are situated chiefly near the cortex, although they may be numerous in the white matter also. Sometimes numerous small cancer nodules may become confluent forming one large mass. The inner portion of the brain may become involved because of the extension of the process by means of the arteries.

Except in the instances in which there is a general carcinomatous process, the spinal cord is rarely involved. Metastasis to the spinal cord is so extremely rare that we have been unable to find a single instance of uncomplicated cancerous metastasis to the substance of the spinal cord, except perhaps in the case of Kolisko, which Schlesinger[2] (p. 82) quotes.

Metastasis into the spinal cord occurs occasionally in conjunction with vertebral carcinomatosis, as in the first case with necropsy recorded by Ballet and Laignel-Lavastine[12], in which a cancerous embolus of a dural artery caused a secondary growth. It is not infrequent to find a diffuse infiltration of cancer cells in the spinal cord in conjunction with meningeal carcinomatosis. Schlesinger also mentions a case of Ewald's in which there was an isolated cancer nodule in the cauda equina. A similar instance is recorded by Scanzoni[13]. In this case, however, there was in addition carcinoma of the vertebræ and multiple carcinomatosis of the brain and cord.

Secondary carcinoma may not only involve the substance of the brain and cord, but also the meninges. In an able paper Siefert[11], in 1903, called attention to this subject and recorded four such cases, besides reviewing the literature. In nearly all of these cases there is dissemination of cancer nodules throughout the brain, and rarely in the cord, invasion of the pia with rapid extension of the cancer cells within the pericerebral and perispinal spaces and though not constant, secondary infiltration of the meninges over the brain, spinal cord and spinal roots.

The extent of the meningeal infiltration varies of course, and depends upon the number and size of the cancerous tumors. Siefert believed that at least in those cases in which the tumors are peripheral and reach the pia, meningeal infiltration is possible. This, however, does not always occur, for cases are recorded in which meningeal infiltration was absent although the tumor mass invaded the cortex and pia.

The meningeal infiltration, according to Siefert, usually begins in the cerebral meninges and rarely in the spinal. Again, spinal infiltration may be entirely absent. The infiltration may be either in the form of a continuous mass or as a flat tumor, or may be in separate masses. The former is the more common. In general it may be said that carcinomatous meningitis resembles greatly sarcomatous meningitis, both in its pathological and clinical peculiarities. Among the authors who have recorded such cases, in addition to Siefert, are Lilienfeld and Bender[14], and Scanzoni[13].

Meningeal infiltration may be present alone, without involvement of the nervous tissues. Such are the cases of Saenger[15], in which there was a pial infiltration at the base or the brain and also in the lateral surface, and of Hellendall[16], in which there was a diffuse carcinomatous infiltration of the cerebral dura.

As is well known, vertebral carcinomatosis is a frequent form of cancerous involvement. We will not discuss this subject extensively, as we[17] have written before about vertebral carcinomatosis, and will only consider the unusual complications. As a rule the cancer cells invade the space between the bone and the dura mater, and exceptionally, the cancerous cells surround the entire dura and the spinal roots. Very exceptionally, the cancer cells penetrate into the dura and more rarely into the spinal cord itself. Such are the cases of Ballet and of Laignel-Lavastine[12] Taylor and Waterman[18] recorded an exceptional case in which the cancer cells invaded the spinal membranes and were entirely within the dura. The infiltration surrounded the whole spinal cord and infiltrated both the anterior and posterior spinal roots, and in the postero-lateral portion invaded the spinal cord.

The rapid involvement of the spinal cord which is sometimes seen in the course of carcinomatosis has always excited great interest. An acute myelitis may occur without the slightest pressure, and is generally explained as a result of intoxication.

Although the primary growth may be a scirrhous cancer, metastatic tumors of the nervous system generally contain more cancer cells and less stroma. On account of growth of vascular connective tissue the tumor may appear papillomatous. The secondary cancer masses found in the ventricles can always be differentiated from the primary growths by the scarcity of connective tissue in the former (Kufs). This was well shown in the case of Kufs.

Within the cancer nodules hemorrhages and areas of softening are sometimes observed. The nervous structures around the cancer growth are only slightly altered, there being perhaps a little glia cell increase, as the nervous tissues are pushed aside and destroyed.

It has perhaps been insufficiently recognized that carcinoma of any organ of the body, but especially of the nervous system, may cause degenerative changes in the nerve cells of the brain and spinal cord at parts remote from the tumor. Whether this is the result of an auto-intoxication, or of a toxin of cancer, or of cachexia, or perhaps of all of these causes, is a difficult question to answer. We have found in our cases in which metastasis occurred either to the brain or to the vertebræ, disease of the nerve cells of different parts of the brain and cord. This subject will be further discussed under toxic changes.

It may be said that the clinical diagnosis of carcinoma of the nervous system is most difficult. The principal reason for this is the multiplicity of the lesions. Most varied diagnoses have been made in cases of carcinoma: brain tumor, hysteria, and syphilis being the most common. Whenever cancer occurs in any of the viscera, subsequent nervous symptoms should cause immediate suspicion of metastasis; but the diagnosis is often difficult because the primary lesion is not detected. Even the diagnosis of the vertebral carcinomatosis may offer difficulties, for as we have here pointed out in a previous communicaton[17], it is sometimes difficult to differentiate between multiple neuritis, spinal caries and vertebral carcinomata.

Alterations in the Nervous System Not Due to Metastasis.

The diffuse degenerations in the spinal cord which sometimes accompany cachexias are well known. Lubarsch[19], for instance. examined the spinal cords of nineteen cases of cancer of different regions and found alterations in eleven. These consisted mostly of a diffuse degeneration of the posterior and lateral columns such as are found in anemias and other cachectic conditions Sometimes the anterior columns may also be partially diseased It is not uncommon to find accompanying these diffuse degenerations, disease of the spinal roots, especially the posterior. and also of the peripheral nerves.

Meyer[20] recorded an exceptional case in which the symptoms of primary disease of the pyramidal tracts appeared, at the same time as the symptoms of a primary cancer of the uterus.

The involvement of the spinal cord may be in the form of an acute myelitis. This may appear independently of any other involvement of the spinal cord, as in the case of Nonne. or may accompany vertebral carcinomata, independently of any compression of the cord.

Clinically the diagnosis of diffuse degeneration of the spinal cord occurring in carcinoma is not always made. This is sometimes because of mild spinal involvement, but mostly because of the preponderance of the other symptoms. It is still a question whether these lesions are the result of a toxin, or merely of a cachectic condition. The latter probably is the more potent cause.

Alterations in the Peripheral Nerves and Muscles.

The peripheral nerves may be involved in three ways. First, by direct contact with the cancer growth, as in a vertebral cancer, or as in Case I. of our series in which the oculomotor nerve was partially involved by the growth in the hypophysis. Second, there may be an acute neuritis due to the cancer toxin, or a chronic inflammation due to the cachexia. Third, the peripheral nerves may be normal, but, as in the case of Raymond[1], the finest intramuscular nerve endings may be implicated by innumerable cancerous masses in the muscles, a true carcinomatous neuromyositis.

The symptoms may come on acutely, as in the case of Francotte[22] (Case 2). In our Case III. the patient had alcoholic multiple neuritis, but in the course of a year most of the symptoms disappeared, although the reflexes remained lost. Later the symptoms of an esophageal cancer appeared, and coincident with this, the neuritic symptoms reappeared, and became gradually more acute. This case illustrates well the influence of cancer toxin on peripheral nerves. Alterations in the peripheral nerves and muscles without any symptoms are common, and are probably caused by the cachexia, as in our Case II. in which the peripheral nerves of one hand were degenerated, although there were no symptoms. Similar instances have been recorded by many authors, as Auché[23], Oppenheim and Siemerling[24], and others. Histologically the alterations are chiefly parenchymatous.

Besides a multiple neuritis, a neuritis of one or a number of cranial nerves or of a plexus, may occur. For instance, in our Case VII. several cranial nerves were paralyzed, while at necropsy, a degeneration of all of these nerves affected clinically was not found. Such alterations are undoubtedly toxic, and will be considered under that head. At times paralysis of a brachial plexus may occur, and this may be caused by the proximity of a cancer of the breast, the cancer poison being carried by the lymphatics.

Changes in the Nervous System Due to Carcinoma Toxin.

Some authors still dispute as to whether the rather bizarre nervous symptoms which may occur in the course of carcinoma are of toxic origin. Saenger, for instance, believes that definite organic changes cause the so-called toxic symptoms, while other authors believe that gastric intoxications are the cause. However, there is no doubt that different symptoms may appear in a case of carcinoma in which at necropsy no macroscopical lesions are found to explain them, but by microscopic examination diffuse changes may be discovered, which are not commensurate with the clinical symptoms.

Such a disease as carcinoma exerts a powerful influence upon the various functions of the body, the intensity of which probably depends upon the nature of the growth plus the inherent resistance of the nervous system to untoward influences. In no other manner can we explain the occasional involvement of the nervous system in carcinoma of other structures.

The nerve cells in carcinoma are particularly susceptible. Thus Sirleo[25] in two different cases of cancer of the abdominal walls examined the nervous system by the different modern staining methods, and was able to demonstrate by the Nissl method a degeneration of the nerve cells of the cerebrum, cerebellum, spinal ganglia and especially of the cells of the anterior horns of the spinal cord. It is probable that if the nerve cells were examined in every case of carcinoma, whether the nervous structures were macroscopically involved or not, degenerative changes would often be found.

In almost all of our cases the nerve cells were more or less diseased, and curiously enough, were much more degenerated in the so-called toxic cases than in those in which the nervous structures were the seat of cancer. The extent of the involvement varies, but it may be said that the greater the toxemia, the more the cells are diseased. The alteration of the nerve cells is always diffuse, and is never limited to certain parts of the brain and cord. Thus in Cases VI. and IX. in which clinical signs were found, the degeneration of the nerve cells was not limited to the bulbar nuclei, but involved other parts. Again in Case VII. in which the clinical symptoms pointed to an involvement of different cranial nerves or their nuclei, the degeneration was not limited to these nuclei, but involved others, although symptoms of this extensive disease were not manifest. Again, nuclei of nerve cells may be normal although from the symptoms pathologic changes may be expected.

As a rule, the changes occurring in the nerve cells are not very intense and consist in a displacement of the nucleus to the periphery, and a disintegration of the chromophylic elements. The nerve cells, however, may be intensely diseased and swollen and their chromophylic elements entirely disintegrated.

Whenever toxic symptoms occur, some of the cranial nerves are nearly always involved as in the case of Miura[26], Bettelheim[27], and in Case VII. of our series. Of the cranial nerves, those concerned with ocular movements, and the optic nerves, are most frequently involved. Microscopically the alterations consist principally in swelling of the axis cylinders, the myelin sheath not being much altered. At times the cranial nerves which show involvement clinically, may not be altered pathologically, and *vice versa.* In one of our cases, No. VII. quite a large area of swollen axis cylinders was found on either side of the aqueduct of Sylvius. This indicates that the cancer toxin causes degenerative changes not only in the nerve cells but also in the nerve fibers, whether in the cranial nerves or in the brain itself.

The blood vessels may be the seat of intense congestion as in Case VII. in which many of the small blood vessels in the cerebrum, optic thalamus, cerebral peduncles and pons were intensely congested, especially near the oculomotor nuclei. In places a round cell perivascular infiltration was found.

Clinically, the toxemia of carcinoma may produce a variety of conditions. Coma, mental apathy, psychical disturbances, hemiplegia, monoplegia, convulsions either general or Jacksonian in type, bulbar symptoms and involvement of the different cranial nerves have all been recorded. In fact it may be said that the toxic symptoms due to cancer may be as varied as the symptoms found in hysteria or syphilis.

We have already written upon bulbar symptoms occurring in carcinomata, but because of additional cases we will again consider this subject. The first case of this kind was recorded by Bruns[28], the second and third by us (Cases VI. and IX. [17],[29], and the last by Warrington[30]. In the case of Bruns nothing was found microscopically, while in our two cases and in the case of Warrington alterations were found in different nerve cells of the bulb, the alterations, however, were not commensurate with the symptoms. In all of these cases the symptoms were purely bulbar, and consisted in atrophy or paralysis of the tongue and palate, difficulty in talking and swallowing, and dribbling of saliva.

In two additional cases which have come under our observation the symptoms appeared directly after operative procedure, in one after an abdominal operation and in the other after removal of a cancer of the glans penis with subsequent sloughing of the part. It seems as if the disturbance of circulation caused by the operation increased the dissemination of the toxic substance. In one case (No. XI.), besides irregular jerkings of the head and limbs, a paresis of one facial nerve and some nausea and vomiting occurred, the patient had difficulty in pronouncing words and stuttered frequently. These symptoms lasted only a short time and improvement occurred rapidly.

In the other case (No. X.), the symptoms consisted in a loss of function in the muscles of articulation, and at times in complete aphonia. The patient tired very easily, especially in the back and head, and when examined three months after his operation, his speech was slow, monotonous but strong, hesitating but not like that of multiple sclerosis. A throat examination did not show any organic changes. There were no other symptoms.

It is rather difficult to explain why these bulbar symptoms should occur except as a result of a general toxemia, when those functions are disturbed first which are most employed.

We have already mentioned that the cranial nerves are frequently involved in cancer toxemia, and that the ocular muscles may be the seat of paralysis. Thus in the case of Miura there was bilateral optic neuritis and paresis of the oculomotor and abducens nerves; in Bettelheim's case, one third and one seventh nerve were paralyzed, while in Case VII. of our series there was complete internal and external ophthalmoplegia of one side besides a facial palsy.

Psychic symptoms are uncommon in the course of carcinoma. Elzholz[31], in 1898, made a résumé of the literature and collected fifteen cases of cancer with psychoses. Of these, in thirteen there was metastasis in the brain, and he concluded that in most cases psychic symptoms occur only in metastasis. They may, however, be of toxic origin, as in Case XI., in which the patient's mind was cloudy and the meaining of remarks made to her was not understood; and in another case observed by us in which a patient with a carcinoma of the uterus, but with no metastasis to the nervous system, had periods of excitement.

The psychoses are generally of the confusional type, but occasionally the patients are maniacal or the symptoms may be those of general paresis (Elzholz).

Case I. A woman, 52 years of age, had a cancer of the breast removed two years before she came under the observation of Dr. James Tyson and Dr. C. K. Mills, to whom we are indebted for the notes and the material of this case. She did not have recurrence in the breast, but numerous nodules, evidently cancerous, developed in different parts of the integument and in the upper part of the scalp.

Since the operation she had not been well and was treated for "nervous prostration" several times. She had dizziness, nausea, and irregular headaches, especially in the left orbit and temple. Her vision gradually became impaired, and the limbs weak. The patellar reflexes were exaggerated.

The eyes were examined by Dr. G. E. de Schweinitz, who reported as follows: "Vision, O. D., 3-30; O. S., 3-60. Pupils: right 2. mm., left, 3. mm. Wernicke's reaction sign present. There is palsy of the left levator and left superior rectus muscles. The ophthalmoscope shows the eye grounds to be gray, slightly blurred discs, but no neuritis nor atrophy in any advanced stage. Bitemporal hemianopsia, as shown below, was present." Dr. de Schweinitz thought that from the ophthalmoscopic standpoint a lesion so situated as to involve the posterior part of the optic chiasm and the superior division of the left oculomotor nerve would explain the symptoms.

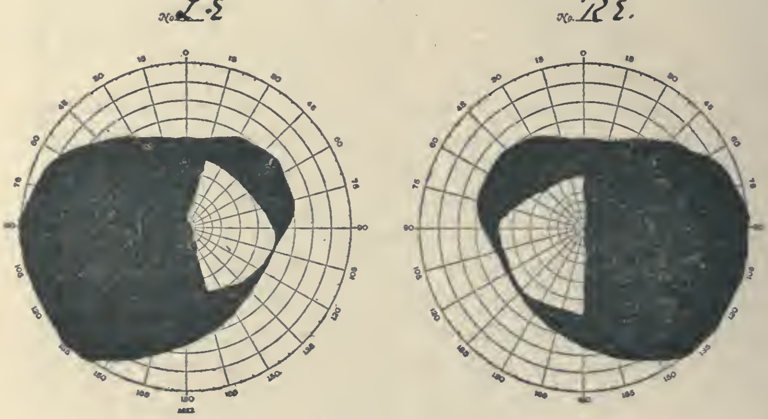

Fig. 1.—Bitemporal Hemianopsia.

The necropsy was performed by us. The examination showed a lump of stony hardness in the exact center of the vertex. On removing the scalp the growth was found to measure one inch laterally and one and one half inches anteroposteriorly. It extended through the calvarium and was adherent to the dura.

The cerebrospinal fluid was not increased in amount, and the brain was not especially edematous. The dura was not adherent to the brain even at the seat of the growth described above.

The sella turcica was much enlarged, being almost one and one half inches in width from side to side, and one inch from before backward. A large tumor mass filled the sella turcica and extended around the optic chiasm and grew into it. It was more extensive on the left side than on the right, and extended as far posteriorly as the anterior part of the pons. The right optic nerve was free from all tumor tissue, the left was attached to the tumor mass.

Scattered throughout the general integument were several hard masses ranging in size from a large hen's egg to a pigeon's egg. The largest of these was within the right scapula. Several smaller nodules were found in the mid-axillary line about the junction of the eighth rib, one in the left groin, and one in the left shoulder. A large tumor mass was also found invading the summit of the uterus.

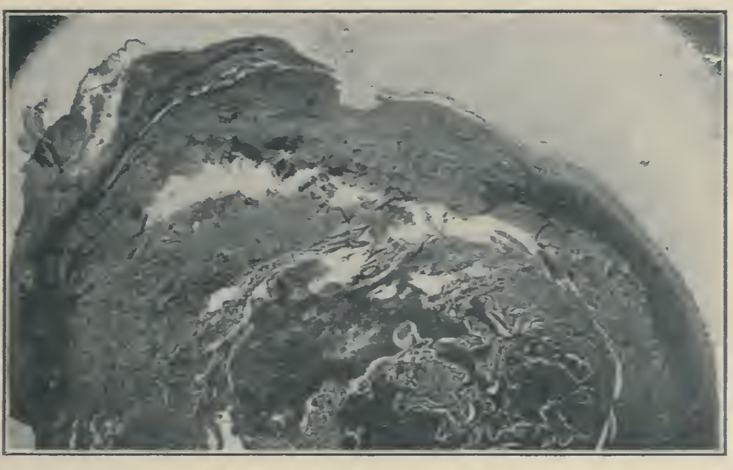

Fig. 2.—Showing Invasion of Chiasm by the Carcinoma; a Few Nerve Fibers of the Chiasm Are Seen on Each Side.

Microscopic examination: The chiasm was much invaded by the carcinoma with great destruction of tissue. The optic nerves did not appear degenerated, or if so, very slightly. A round cell infiltration was found in the pia of the medulla oblongata. A section of the left oculomotor nerve showed invasion by carcinoma cells, and the nerve was adherent to the tumor mass.

Case II. A woman of about sixty, was a patient of Dr. M. B. Dwight, and was seen by one of us with Dr. Dwight and Dr. Wm. Evans. She had had a tumor removed from the right breast twelve years ago, and a few months before she was seen in consultation had had another operation when some tissue, probably carcinomatous, was removed. She was well until eight months before coming under observation, when she complained of pain in the back of the neck, and this region was also tender to pressure.

Examination showed the pupils to be of equal size, the reaction to light was normal. Extraocular movements were normal. There was no involvement of any of the cranial nerves. Sensations for touch and pain were normal over the face.

The patient could not move the right upper limb at the shoulder, but all of the other movements of this limb and of the left upper limb could be performed slowly. Grasp of each hand was feeble, and she had difficulty in picking up a pin. The biceps and triceps tendon reflexes were about normal.

The lower limbs were spastic, and when walking the steps were short, but she did not stagger. The patellar jerks were equally exaggerated, but there was no ankle clonus. The Babinski reflex was uncertain. Sensation was diminished in all four limbs for touch and pain, pain sensation being more diminished in the right upper and left lower limbs. There was no pain on pressure in any of the limbs.

The necropsy was performed by one of us. The carcinomatous mass involved the vertebræ and the surrounding structures from the third to the sixth cervical vertebræ inclusive, rendering the tissues soft. The spinal cord was compressed in this area; the area of softening beginning in the lowest portion of the third cervical segment and involving the fourth, fifth and upper portion of the sixth cervical segments. The carcinoma tissue invaded the dura but did not penetrate it.

Microscopical examination: There was no very distinct degeneration by the Marchi method, either in the cervical or thoracic regions of the cord, although a few black dots were found in the crossed pyramidal tracts. These black dots were not very numerous but could be regarded as a slight secondary degeneration. No degeneration was evident either in the cervical or lumbar regions by the Weigert hematoxylin, or the acid fuchsin hemalum stains. With the thionin stain, some of the cells of the anterior horns were diseased but the alterations were not very distinct. The paralysis in greater part must have been due to a compression of the cord, which was not sufficient to cause intense secondary degeneration.

In the medulla oblongata the Marchi stain showed intense degeneration of the columns of Burdach on each side. The Weigert hematoxylin and acid fuchsin hemalum stains did not show any pathological changes. The cells of the nucleus ambiguus and of the twelfth nucleus were normal.

The Betz cells of each paracentral lobule showed some alteration, the cell body being swollen and the nucleus displaced; there was also considerable disintegration of the chromophylic elements.

A piece of muscle from one hand did not show any degeneration but a piece of nerve showed slight degeneration by the Weigert hematoxylin method.

This case was clearly one of vertebral carcinomatosis, but clinically the intense pains which usually accompany this disease were absent. The pathological findings were not exceptional except that the nerve cells of the anterior horns in the spinal cord and especially the Betz cells of the paracentral lobules were degenerated. This probably was caused by the cancer toxin, as was also the degeneration found in a peripheral nerve, of which no clinical signs had been manifest.

Case III. W. McF., was admitted to the nervous wards of the Philadelphia General Hospital, December 8, 1903. This patient was a man of sixty years and had alcoholic multiple neuritis of the ataxic form, and had in addition the psychosis frequently seen in this disease. He recovered after a long illness and about a year later the only remaining symptoms were those of lost tendon reflexes, slight steppage gait and perhaps a little tenderness on pressure over the nerve trunks of both feet. His mentality at this time was normal.

About one year after the subsidence of the symptoms of multiple neuritis he began to complain of difficulty in swallowing and regurgitated food. Gradually these symptoms increased, until he was unable to swallow anything but liquids. His strength failed, and he became cachectic. An esophageal cancer was diagnosed. Coincident with the appearance of these symptoms he began to complain of pains in his limbs and of great tenderness on pressure over his nerve trunks. These neuritic symptoms gradually grew worse, and became almost as severe as in the previous attack.

He died January 30, 1906. An esophageal cancer was found.

Microscopically, the spinal cord was normal. The nerve cells of the anterior horns were in fair condition. A piece of nerve of one hand showed very intense chronic degeneration, the nerve fibers had almost entirely disappeared. A piece of muscle from the same hand showed considerable atrophy of some of the muscle fibers, with overgrowth of the connective tissue. A piece of nerve teased in the fresh state and stained by osmic acid showed no recent degeneration.

This case illustrates the effect of carcinoma toxin upon peripheral nerves, as shown by the reappearance of the neuritic symptoms.

Case IV. Admitted to the Philadelphia General Hospital, March 13, 1906. The patient was a woman of about 40 years of age who, one year before she came under our observation had a cancer of the left breast removed. About ten months after this she began to complain of radiating pains across the chest and back, and in her lower limbs. A month after this she suddenly lost power in her lower limbs, since which time she has been in bed. She complained of girdle sense but not of extreme pain. Examination showed the cranial nerves to be normal. Her mentality was good. The upper limbs were not involved. She was unable to move her lower limbs and these were in flexor contracture, the legs on the thighs and the thighs on the abdomen. The patellar and Achilles jerks were not obtained on either side. The Babinski response was positive on both sides. Sensations for touch and pain were lost over the lower limbs, abdomen and chest to a point about three inches above the umbilicus, except on the dorsum of each foot, where occasionally a pin prick was recognized. There was incontinence of urine and feces. The diagnosis of vertebral carcinoma was made. The patient died, April 3, 1906. The necropsy showed a carcinomatous involvement of the thoracic vertebræ with a compression of the upper thoracic chord.

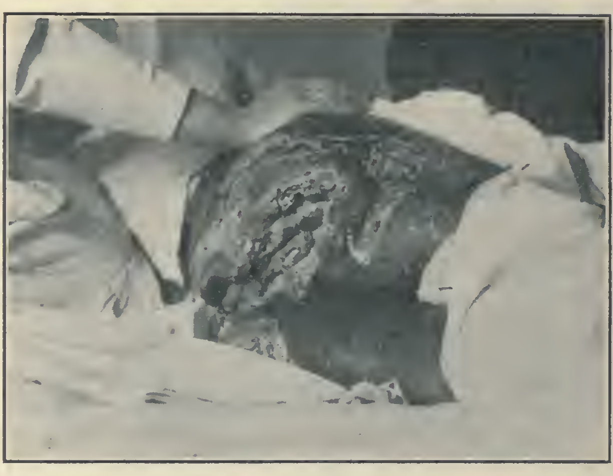

Fig. 3.—Intense Bedsore Occurring with Vertebral Carcinoma.

Case V. Beard, was admitted to the nervous wards of the Philadelphia General Hospital, December 24, 1904. He denied venereal disease. The history obtained was very unsatisfactory.

The face and upper limbs were not affected, but the lower limbs were almost completely paralyzed, although some movement was possible at each hip and each knee, and the toes were moved slightly. The patellar reflex was exaggerated on each side, but ankle clonus was not obtainable. Babinski's reflex was distinctly present on each side. Sensations for touch and pain were preserved in the lower limbs, but tactile sensation was diminished on the soles of the feet. Retention of urine was present.

Dr. Knipe reported, January 10, 1906, that the patient had paresis of the left external rectus and of both superior recti, nystagnoid movements on extreme right lateral deviation, severe grade of optic neuritis (partially choked disc) in the right eye, with atrophied spots above following old hemorrhage, and a few fresh hemorrhages near the disc to the inner side. The disc of the left eye was markedly swollen, but not so much as that of the right eye.

There is only one record of any pain in the lower limbs and this was made July 10, 1905.

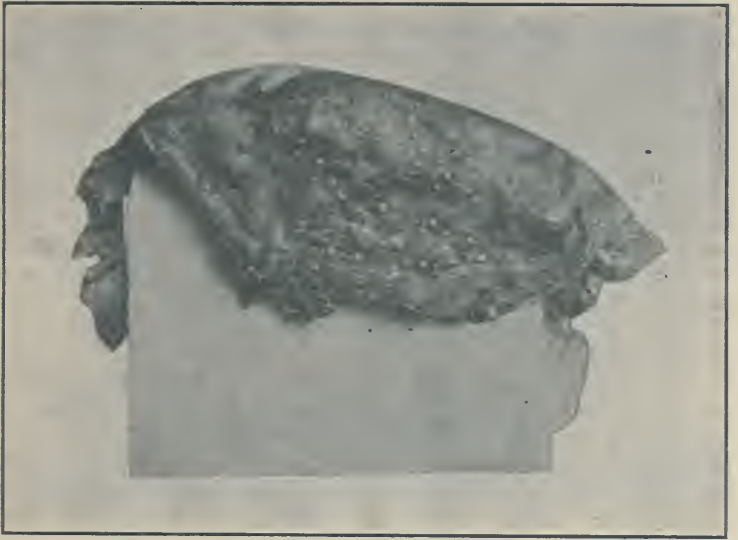

Fig. 4.—Numerous Carcinoma Nodules from the Size of a Millet Seed to that of a Pea on the Inner Surface of the Cerebral Dura.

He came again into the service of one of us in January, 1906. At that time his condition was as follows: He lay in bed with the thighs strongly flexed on the abdomen and the legs flexed on the thighs. He had slight voluntary power in flexion of each thigh, but it was very doubtful whether he had any voluntary movement of his toes. The slight upward movement of the toes which sometimes occurred was probably reflex. The lower limbs were much wasted. The patellar reflex and Achilles tendon reflex were absent on each side. The Babinski reflex was very typical on each side. Tactile and pain sensations were entirely lost in the lower limbs. He had no control of the urine or feces, and the bowels did not move without enema. He moved the upper limbs freely but the movements were weak. These limbs were also wasted. Biceps and triceps tendon reflexes were present on each side, and about normal, considering emaciation which was general. The pupils were equal. The movements of the eyeballs were probably good, though it was impossible to get him to respond promptly. No impairment of cranial nerves was detected. The abdominal muscles were intensely rigid and the abdomen was distended. A necropsy was obtained.

The bone on the whole left side and upper portion of the skull was thickened, and measured about ¾-inch in thickness at the mid portion. The thickening seemed to be caused by the infiltration of a tumor connected with the dura and covering internally the thickened portion of the skull. The tumor (carcinoma) invaded the posterior portion of the second, third and fourth left temporal convolutions; its upper margin began at the lower portion of the second temporal convolution and the anterior margin at the mid portion of the affected convolutions. It involved the tentorium posteriorly. The dura covering the left cerebral hemisphere was thickened and numerous nodules from the size of a millet seed to a pea were found on the inner side of the thickened dura. These were shown to be carcinomata. The spinal dura was adherent at the sixth, seventh and eight thoracic vertebræ, and the bodies of these vertebræ were soft. Carcinomata were found in the liver, ribs and prostate gland.

The absence of all cerebral symptoms, except ocular, in this case was remarkable.

Case VI. This case has been previously reported by us, and only an abstract is given. M. R., a woman, forty-eight years old. Eight years before coming under observation had an abscess(?) of the right breast which ruptured and discharged pus. Four years afterward she began to have sharp shooting pains in her back and around her waist, and later pain in her limbs. Since three weeks after the beginning of her pains she had been confined to her bed. There was extreme atrophy in all of her limbs, more marked in the lower. The upper limbs could be but weakly moved voluntarily. No voluntary movement was possible in the lower limbs. The arm reflexes were present and exaggerated but the reflexes of the lower limbs were absent, probably on account of the extreme atrophy. Sensation was irregularly diminished. She had sharp, spontaneous pains in all of her limbs, and the slightest touch would cause extreme pain. The lower limbs were contracted in flexion. There was a bilateral atrophy of the tongue with fibrillary tremors and weakness in movement.

At the autopsy, metastatic carcinomatous growths were found in the cervical region implicating the bodies of the vertebræ and the surrounding structures, and in two places the growths had implicated the spinal canal. These growths were on the outer surface of the dura and did not penetrate the membrane. The spinal cord was compressed.

In the miscroscopic examination, the carcinomatous masses found outside of the dura, did not penetrate very far into the dura, and distinct carcinomatous tissue was not seen within this membrane. The carcinomatous masses were chiefly on the posterior part of the dura. The spinal cord showed the usual changes of compression myelitis.

In this case we have the typical symptoms of a vertebral carcinomatosis. The bulbar symptoms were unusual.

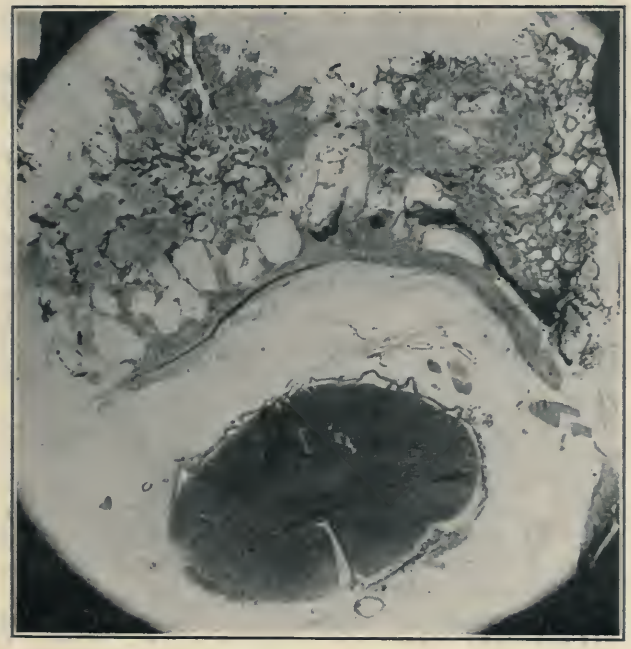

Fig. 5.—Carcinoma on the Posterior Part of the Spinal Dura Not Penetrating the Dura.

Case VII. Was admitted to the Philadelphia General Hospital, September 8, 1903, in the service of Dr. H. B. Allyn, but was transferred to the service of Dr. C. H. Frazier because of fracture of the humerus. The fracture occurred while she was being lifted in bed to an upright position. The notes then made were as follows: "The patient is fifty years of age. A carcinoma of the breast was removed sixteen months before she came under observation, and she has a recurrence in the same place. She complains of pain in the back of the neck and in the hips, especially the left, at the site of operation, and in the back. Five weeks before her admission to the hospital, coincident with the pain in the neck she began to have failure of vision. For several months she has had morning vomiting, and about a year ago she had a swelling at the carpo-metacarpal joint of the left thumb.

There is some exophthalmos of the right eye, but no ptosis. The right pupil is smaller than the left, and responds to light and in accommodation. The right external rectus is paralyzed. She says she can see better with the left eye.

There is ptosis of the left eyelid. The left pupil is larger than normal and does not react to light or in accommodation. The left pupil does not respond to pain stimulus, whereas the right pupil does. Paralysis of the left external rectus and partial paralysis of the left superior rectus exist.

There is no paralysis of the other cranial nerves, nor involvement of the special senses other than that of sight. Both hips are tender and she cannot stand without support."

Other notes made later by one of us (Dr. Spiller) were as follows: "The patient has almost complete ophthalmoplegia, internal and external, of the left eye, and distinct but not equal weakness of the left seventh nerve. She complains of headache over the frontal region, and has had dizziness and weakness of the legs. The patellar reflex is lost on each side, and there is no ankle clonus or Babinski response on either side. She has marked tenderness over the spinal column between the shoulders but no deformity of the spinal column or tumor formation." She died three weeks after her admission to the hospital.

Macroscopical examination did not show any gross changes. The microscopical examination was as follows:

Cranial nerves.—The optic nerves were perfectly normal, both by the acid fuchsin and Weigert hematoxylin stains. A cross section of the optic chiasm did not show any pathological changes.

Some of the axis cylinders of the oculomotor nerves of each side were distinctly swollen.

The left fourth nerve was normal, but some of the axis cylinders of the right fourth nerve were distinctly swollen, but not so much so as those of the third nerve.

The right sixth nerve showed intensely swollen axis cylinders by the hemalum acid fuchsin stain. The left sixth nerve was not obtained.

Some of the axis cylinders of both seventh nerves were swollen, but not nearly so much as those of the right sixth.

Medulla Oblongata.—Sections from the medulla oblongata seemed to be normal. The blood vessels were not congested.

Pons.—The blood vessels within the pons were much congested, and this congestion was more distinct in the posterior part of the pons. There was also some perivascular round cell infiltration. Quite a large area of swollen axis cylinders was found on each side of the aqueduct of Sylvius.

Cerebral Peduncles.—A similar state of congestion was found in the posterior part of the cerebral peduncles, especially around the nuclei of the oculomotor nerves.

Optic Thalamus.—Sections from this area showed intense congestion, there was also a slight perivascular infiltration around the blood vessels.

Cortex.—There was considerable congestion in sections from each frontal and each occipital lobe, but not nearly so much as in the pons and cerebral peduncles. The cerebellum was normal.

Nerve Cells.—Many of the cells of the third nerve on each side were distinctly diseased, being swollen, presenting chromophylic changes involving all parts of the cell, and the nuclei were displaced to the periphery. Many of the cells of these nuclei were normal.

The sixth nucleus on each side was very much affected, if anything more so than the oculomotor, and in a similar manner.

The seventh as well as the twelfth nucleus on each side was perfectly normal. The nucleus ambiguus of one side, the Betz cells of each paracentral lobe, and the nerve cells of each occipital and frontal lobe were in a healthy condition, but some of the cells of Purkinje showed some displacement of the nucleus.

The symptoms in this case were probably caused by the carcinoma toxin. This case illustrates well the widespread effect of such a condition, and the pathological changes found in the nerve cells, blood vessels and cranial nerves are typical.

Case VIII.—A woman about 60 years of age, was seen in consultation by one of us with Drs. L. W. Steinbach and Dr. David Riesman. Two years before this examination a carcinoma of the left breast was removed by Dr. Steinbach, and for the past fifteen months she has had pain and swelling in the left knee, and pain in the neck for about seven weeks.

There was no cachexia and intellect was clear. Vision in the right eye was good, in the left eye a cataract operation had been performed. The light reaction in the right eye was prompt, all the cranial nerves were normal.

The movements of the neck were restricted in all directions. She could move her head from side to side and backwards and forwards slightly, but the movements caused pain. Turning her in bed caused excruciating pain. There was a swelling in the spinal column in the back of the neck.

It was impossible for her to move the left upper limb at the shoulder, but she could flex the left forearm on the arm freely. The grasp of each hand was normal. Movement in the right upper limb was good. The biceps and triceps tendon reflexes were exaggerated on both sides, much more so on the right side. Sensations for pain and touch were normal everywhere.

Much numbness and pain were present in the left upper limb.

There was no voluntary movement in either lower limb, except in the toes where movement was free. The left thigh seemed smaller than the right. The patellar reflexes were exaggerated, more so on the left, as shown by the prompter contraction of the quadriceps, rather than by movements of the leg. The Achilles jerk was not distinct on either side, and the Babinski response was not obtained, the toes not moving on plantar irritation. Any movement of the lower limbs caused intense pain. There was no tenderness to pressure over the spinal column.

This patient also showed the usual symptoms of vertebral carcinomatosis. She subsequently died, but an autopsy was not permitted.

Case IX.—This case has been reported by one of us in a previous communication and only a short abstract will be given. A woman of fifty-nine years developed a cancer of the right breast thirteen years previously. Five months before her death she began to complain of difficulty in swallowing and talking. When examined a month before her death she could talk only in a whisper and could swallow fluids only with difficulty. There was no atrophy of the tongue, lips or face.

At the autopsy no macroscopic lesions were found, but microscopic examination showed an alteration in some of the nerve cells of the nuclei of the sixth, seventh, ninth and tenth cranial nerves.

This was the third case of bulbar symptoms occurring in carcinoma reported, and the first in which bulbar symptoms occurred alone. These were not due to an alteration of some of the nerve cells, but to the general intoxication.

Case X.—A man of forty-one years, with an excellent family and past history, was sent to one of us for opinion by Dr. W. M. Robertson, of Warren, Pa., with the following history: Three months before he came to Philadelphia he was seen by Dr. Robertson, the patient then having a cancer of the glans penis, following phimosis. A few days later the part was amputated and the inguinal glands removed. Several days after this he developed a cystitis and urethritis from the retention catheter, and at the same time the end of the stump sloughed. These symptoms caused a high temperature for a few days.

The condition of the patient was good during the fever, but when it left, there was loss of function of the muscles of articulation and at times complete aphonia. The mental condition was good. He also had some photophobia and double vision. After a week his voice returned somewhat but did not fully recover its former quality. Examination by one of us three months after the onset of the cancer symptoms showed the pupils to be normal in size, the reaction to light, accommodation and convergence normal. Extraocular movements were not impaired. The facial and trigeminal nerves were normal on either side. Sensations for touch and pain were normal over the face.

The upper limbs were not weak, but there was not much resisting power. There were no subjective pains. Sensations for touch, pain and temperature were normal in the forearms and hands. The biceps and triceps tendon reflexes were normal. Power in the lower limbs was normal. The patellar and Achilles jerks were normal. The Babinski sign was not obtained. Sensations for touch, pain and temperature were normal over the feet.

The voice was slow, monotonous but strong; hesitating but not like that of multiple sclerosis. The patient would tire easily, especially in the back of the head and neck. After an automobile ride he became extremely exhausted and had complete aphonia. Weakness was so great that he could not turn in bed. All voluntary muscles moved slowly.

Dr. Freeman examined the throat and reported that there was no organic disease of this part. There was some congestion of the larynx and trachea which made speaking a little difficult and caused the patient to clear his throat, but Dr. Freeman did not believe that this had any influence on his curious manner of speaking. The tissues of the larynx were relaxed and there was a marked sluggishness in the action of the laryngeal muscles. Dr. Freeman considered the difficulty in speaking as caused by mental disturbance and believed that time would bring about a full recovery.

In this patient the weakness of the muscles of articulation may have been caused by the cancer toxin, and was really part of the general weakness with which the patient suffered. It is a striking fact that these symptoms did not come on until after the removal of the growth and the appearance of sloughing of the part. This can probably be explained as in the following case, by the dissemination of the toxin by the circulation. Some of the features suggest myasthenia gravis.

A letter received from Dr. Robertson, February 12, 1906, is as follows: "Mr. E.—— has done quite well. His improvement is slow but constant. He has grown fleshy. I have been a little in doubt as to whether or not there is present a certain caste of mind like that of senility. The speech is not right, but is better than when you saw him. I can see no evidence of the original trouble being present."

Case XI.—Was seen by one of us in consultation with Dr. Marie Formad. A short time before the examination a carcinoma of the uterus and ovaries had been removed by Dr. Formad. Numerous minute carcinomatous nodules were found in the peritoneum and could not be removed.

A short time after the operation she began to have constant headache, either in the right or left side of the forehead. She also vomited considerably and had occasionally dizziness.

When examined she understood all that was said to her, but at times her mind seemed a little cloudy and she did not grasp immediately the meaning of a remark made to her. She was not aphasic, although at times she had difficulty in pronouncing the word she wished to say and frequently stuttered. Her enunciation was distinct. She could count fingers correctly with each eye, but she acted as though her vision were not entirely normal.

The pupils were equal and the iritic response to light prompt in each eye. Extra-ocular muscles were normal. There was no hemianopsia. There was a slight weakness in the distribution of the right facial nerve as shown by the incomplete elevation of the brow and of the corner of the mouth on that side. The masseter contracted well on each side and hearing was good. There was no rigidity of the neck and movements of the head were free.

The upper limbs were well developed and power was about normal. The biceps and triceps tendon reflexes were prompt on each side, probably a little prompter on the right side. The tonicity of the muscles was about normal.

The lower limbs were distinctly weak. The patient could perform all movements, but had little power if any resistance was offered. The tonicity of the muscles was about normal. The patellar reflex was exaggerated on each side, and there was a tendency to patellar clonus on each side; but the clonic movements were abortive and soon ceased. The Babinski reflex could not be obtained because the patient was very ticklish on the soles of the feet. There was no atrophy of the limbs.

Sensations for touch and pain were normal all over the body. Constant involuntary jerkings of each side of the face were present during the examination, jerkings were present also in both the upper and the lower limbs, but not so constantly as in the face. Gait and station could not be tested because the operation had been too recent. The patient improved considerably under treatment.

The symptoms in this case appeared after the operation, and it is probable that the disturbance of circulation caused by the operation increased the dissemination of the toxic substance from the numerous carcinomatous masses remaining in the peritoneum.

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CEREBRAL DECOMPRESSION.

Palliative Operations in the Treatment of Tumors of the Brain, Based on the Observation of Fourteen Cases.[1]

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REMARKS BY DR. SPILLER.

Palliative operations in cases of brain tumor are not new, and the temporary benefits to be derived in this way have been fully recognized by many physicians during many years. It has seemed time well spent to examine some of the reports of cases treated by trephining, or other method of opening the skull, for the relief of pressure when a tumor could not be localized, or if localized could not be removed. Unquestionably many cases have been omitted, and yet I have collected sufficient to demonstrate that palliative treatment has an important place in cerebral surgery.

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[1] Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Fifty-seventh Annual Session, June, 1906, and published simultaneously in the Journal of the Association.

REVIEW OF LITERATURE.

Annandale[1] said, in 1894, that more than twenty years previously he had opened the skull of a patient whose symptoms were those of general brain pressure and, although no special condition was found, temporary relief was marked. Another case of this kind, he says, was operated on by Sir Joseph Lister in the same wards with success. In 1889, Annandale removed a piece of bone from the skull becaus of intense headache, following an injury of the head years previously. No abnormal condition was found, although the dura was incised. The patient made an excellent recovery and was perfectly cured. Annandale remarked that when no localized symptoms of brain tumor exist an exploratory operation may be performed, and experience has shown that in some of these cases the operation has been useful in taking off general pressure and in a few instances has even been followed by shrinking or degeneration of the growth. Again, a growth may give rise to effusion of fluid or to hemorrhage, and the symptoms caused by these conditions may be at least temporarily removed by the operation.

Sahli,[2] in 1891, spoke of improvement from palliative operations. The tumor in one case was supposed to be in the cerebellum. Vision improved and headache disappeared after a palliative operation. The patient (a boy) died some months later and no necropsy was obtained. Sahli thinks that two trephinings remote from each other give more relief of pressure than one. Sometimes Nature performs a palliative operation, what he calls *Selbsttrepanation*. In consequence of the pressure the skull becomes thin at the region of the Pacchionian bodies and openings may occur, and in one case he saw the brain protruding through several such openings.

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[1] Edin. Med. Jour., January to June, 1894, p. 898.

[2] Volkmann's "Sammlung klin. Vorträge, Innere Medicin," Nos. 1 to 29, 1890–1894, p. 300.

The palliative operation, he thinks, is not ideal, is not without danger, and improvement is only temporary. In one case of prolapse of the brain he observed unconsciousness immediately followed by death. Acute prolapse of the brain may impair the function of the rest of the brain. He recommends small trephinings in preference to large.

In 1891, White[1] described a number of cases of epilepsy in which improvement followed operation, although nothing abnormal was found.

In 1893, Horsley[2] said that the removal of excitatory symptoms by opening the skull is a marked phenomenon, convulsions ceasing. In one of his cases in which the diagnosis of tumor of the middle cerebellar peduncle was made the skull was opened only to relieve the pressure, and the cerebellum protruded almost an inch through the trephine hole. The symptoms of tumor soon disappeared, and six months later the man felt perfectly well. He survived eighteen months, and during this time had few attacks. Instead of a tumor only a small cavity was found.

Horsley thinks that some tumors are so interfered with in their nutrition by opening the skull and suddenly altering the pressure therein that they forthwith degenerate. In another of his cases the tumor was too large for removal, but the patient survived for over two years, with disappearance of the attacks and improvement of the hemiplegia. The tumor at the time of the operation was obviously malignant and rapidly growing, but had later undergone complete destruction by the mere exposure at the time of operation. Such a result, he says, cannot be anticipated or expected as likely to follow, but it is an argument in favor of opening the skull for the purpose of palliating a condition which cannot be cured.

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[1] Annals of Surgery, August and September, 1891.

[2] Brit. Med. Jour., 1893, No. 2, p. 1365.

Horsley says, also, that he has found in every case the effect of opening the skull has been to remove the headache, and, further, that in cases where it was known before operation that the tumor could not be removed, relief from severe pain afforded by opening the skull persisted until the patient died. The simple and safe procedure of opening the skull should be undertaken when drugs fail. At the Berlin Congress Horsley pointed out that opening the skull caused the swelling of the optic disks to subside, and that the subsidence may proceed steadily to complete recovery, provided atrophy has not previously begun. If atrophy has commenced the condition is hopeless. Vomiting is dependent on the degree to which intracranial tension is raised, and, as a rule, this symptom disappears when the skull is opened.

Jaboulay,[1] in 1893, obtained good results by trephining in a case of tubercle of the brain without opening the dura. The prognosis from trephining, he thinks, is better the more superficial and circumscribed the lesion is.

Caton and Paul[2] trephined the skull in a case of acromegaly in 1893. The dura bulged in the opening. The operation relieved the headache, and the remaining three months of the patient's life were comparatively comfortable.

Macewen[3] did a palliative operation in 1889 in a case with symptoms of cerebellar tumor. The patient experienced great relief. After a week tuberculous tumors were removed from the cerebellum.

Taylor[4] reported a series of cases of brain tumor to show that there is almost invariably a subsidence of optic neuritis after operation whether the tumor is removed or not. This fact, he says, was known to Horsley so long ago as 1888. In discussing Taylor's paper Horsley said that if there is any atrophy present no amount of reduction of pressure within the skull will produce the slightest change or afford the slightest relief in respect of the loss of vision. If there be commencing atrophy after operation the neuritis that may be present will disappear, but the atrophy is permanent; opening the skull, therefore, is not justifiable if the atrophy is well marked. Horsley was probably referring to the operation only in so far as vision is concerned, as the operation may relieve some of the other symptoms, even though it may be too late to restore vision. The optic neuritis may disappear, Horsley says, even though the dura is not opened.

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[1] Arch. provinc. de Chir.,1893, vol. ii.

[2] Brit. Med. Jour., Dec. 30, 1893, p. 1421.

[3] Ibid., Dec. 23, 1893, p. 1367.

[4] Transactions of the Ophthalmological Society of the United Kingdom, 1893–1894, vol. xiv, p. 105.

In a case reported by Keen,[1] in 1894, the diagnosis of intracranial tumor was made, but the growth could not be located. Trephining was recommended with a view of relieving intense headache. The tumor could not be found.

The bone was not replaced, and, although the brain bulged greatly, headache and hallucinations entirely disappeared, and the man became a quiet and tractable patient. He died four and a half months after the operation, having been much relieved during this time. The operation was performed in 1892. In another case, also, much benefit was obtained by a palliative operation.

Bruns,[2] in a case of brain tumor in which the tumor could not be found at operation, improved the patient's condition by opening the skull. By the end of the third week very little choked disk remained, attacks of blindness no longer occurred, headache was much less severe and vomiting ceased. Similar improvement he had noticed in two other cases of brain tumor after trephining.

Byrom Bramwell,[3] in 1894, said that he had advocated trephining for the relief of symptoms in his book, *Tumors of the Brain*, published eight years previously, but that he would advocate the operation still more strongly in 1894 in cases in which there is a great increase of intracranial pressure and drugs have failed. When he published his book he had not been aware that Grainger Stewart or anyone else had recommended the operation. In many cases the headache is intense, and it has been shown that in some of these cases sudden death occurred, apparently as a result of the mere severity of the pain and sudden inhibition of the action of the heart. In other cases in which the intracranial pressure is greatly increased, the patient dies suddenly in an epileptic fit or as the result of failure of respiration. Opening the dura reduces intracranial pressure and relieves headache and other symptoms, among them optic neuritis.

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[1] Amer. Jour. of the Med. Sciences, January and February, 1894.

[2] Neurolog. Centbl., 1894, p. 20; also 1893, p. 389.

[3] Edin. Med. Jour., January to June, 1894, p. 1067

In 1894, Kammerer[1] reported a case of supposed tumor of the brain in which double trephining was done for the relief of pressure. The headache disappeared, vomiting ceased for several weeks and then occurred occasionally. The motive power of the right side (previously this side was paretic) improved and some sensation returned. The dura was not incised.

Wyeth remarked that he had trephined in two cases with symptoms of brain tumor, and, finding no tumor, had sewed up the dura, but had not replaced the bone; improvement had followed the relief of pressure.

Sänger,[2] in 1894, reported a case of brain tumor in which after opening the skull and dura prolapse of the brain with improvement in the symptoms occurred.

In one of the cases of tumor reported by E. Albert,[3] in 1895, in which palliative trephining was done, headache disappeared, but vomiting persisted. The dura probably was not opened. Later trephining was done on the other side of the head, but vomiting persisted. The relief afforded in this case was in regard to headache and might have been greater if the dura had been opened.

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[1] Annals of Surgery, January to June, 1894, vol. xix, p. 685.

[2] Neurolog. Centbl., 1895, p. 472.

[3] Wien. med. Woch., 1895.

In another case in which trephining was done for brain tumor the dura was punctured. Headache became at once less severe, but the improvement was only temporary. In another case after trephining apparently without opening the dura, vision improved and headache and vertigo disappeared. The symptoms returned and the dura was opened. The operation was not very successful as a palliative means.

Schlesinger,[1] in a case of brain tumor, had a large protrusion of the brain as a result of the operation. The protruded portion could not be replaced, nor was it excised, and when the patient was presented he had a subcutaneous tumor on his head as large as a fist. The general condition improved as a result of the operation, the palsy diminished, the Jacksonian convulsions ceased on the third day after the operation, and the choked disks and headache disappeared. Seven weeks had elapsed since the operation at the time of presentation. A later report of this case states that the improvement lasted several months and then the symptoms returned rapidly and death soon resulted. The entire right cerebral hemisphere was replaced by a gliosarcoma.

Decompressive trephining, a term employed by Jaboulay,[2] performed for brain tumor, according to this author, has proven to be of some benefit, more often not. When done for the relief of inoperable tumors the results are not always brilliant. He speaks of astonishing results produced by trephining in the cerebellar fossa in a case of basal tumor; the pain ceased and the vision improved. The patient lived four and a half months after the operation. Jaboulay seems to believe that the relief in palliative operations is greater for tumors of the cerebellum and adjoining parts than for tumors of the cerebrum.

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[1] Neurolog. Centbl., 1895, p. 702, and 1898, p. 974

[2] Lyon Médical, 1896, p. 73.

Broca and Maubrac[1] ascribe to Horsley the honor of being the first to describe carefully trephining as a palliative operation in brain tumor, although scattered references to the subject are found in the literature, and they refer to Annandale and Lister. Horsley recommended making a large opening in the skull without opening the dura. Sahli, they say, recommended excision of the hernia that is produced by opening the dura, especially as the portion of the brain involved in the hernia is impaired in its function. They say that Kocher did operate in this way on a child in the service of Sahli for a tumor not localized, but probably in the cerebellum. A hernia of the cerebellum was excised and benefit resulted. Broca and Maubrac employ the term "cerebral decompression." The palliative operation, they think, exposes the patient to the danger of shock, and when the operation is performed at an advanced period of the disease death is not exceptional. When the patient survives the most distressing symptoms are lessened or disappear entirely, and, therefore, the palliative operation should be employed. It does not cause more rapid growth of the tumor. Exploratory trephining, when the diagnosis of location has been incorrect, has taught us that cerebral decompression may give important results. These authors express themselves in favor of partial removal of a tumor when the whole cannot be excised, but are undecided as regards Horsley's view that partial ablation may retard the growth of the remaining portion. They report two cases of tumor in which palliative operation was beneficial.

In 1895, J. J. Putnam[2] remarked that he knew of one patient who, in consequence of simple opening of the skull, had been free from pain for many years, though blind. He said he had seen relief from pain by large openings in the bone in several cases. He was speaking of brain tumors.

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[1] Arch. Gén. de Médecine, February, 1896, p. 129.

[2] Boston Medical and Surgical Journal, January 16, 1896, p. 66.

Schultze's[1] results from palliative operation in brain tumor were disappointing as recorded in his paper published in 1896. He speaks of it as often dangerous, but at times beneficial.

Ludwig Bruns[2] thinks that when the tumor cannot be removed or only a small part of it is removable, trephining does not affect the focal symptoms, but causes the general symptoms quickly to disappear if the opening is large. Consciousness returns, headache, vomiting, and choked disks disappear. In two of his cases the patients, who had hovered between life and death, had been able to walk again after palliative operations. In one of his cases (glioma of the occipital lobe) improvement lasted several months; in a second case it lasted some weeks. As headache in many cases does not return at all the recurrence of symptoms is not so distressing and the patient passes into coma. The tumor, by growing through the opening, removes the pressure on the lymph tracts within the skull. Bruns says the opening in the skull must not be too small, and the dura also must be opened.

Clarke and Morton[3] report a case of tumor of the brain without localizing symptoms, in which a large area of the skull was removed for relief of intracranial pressure and the dura was not incised. Headache disappeared. They remarked that Beevor and Ballance thought that removal of bone alone without incision of the dura was not likely to relieve intracranial pressure, but they (Clarke and Morton) believe that the actual increase in the bulk of the cranial contents may not be considerable if the tumor is small, and the bulging of the dura mater even to a very limited extent may be sufficient to relieve the increase of the intracranial pressure caused thereby. Clinical evidence in Caton and Paul's case, as in their own, showed distinctly that removal of an area of bone alone is sometimes sufficient. Except for the recurrence of fits five months after the operation, the trephining was of great benefit in Clarke and Morton's case. Optic neuritis subsided, leaving some consecutive atrophy, but the patient had good vision, was entirely free from headache and vomiting, except once during the last series of fits, had recovered his mental powers and had a comfortable existence.

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[1] Deuts. Zeit. f. Nervenhlk., 1897, vol. ix, p. 217.

[2] "Die Geschwülste des Nervensystems." S. Karger, Berlin, 1897, p. 229.

[3] Brit. Med. Jour., 1896, p. 802.

Alfred Wiener[1] reports a case of irremovable tumor. After opening the skull, headache and convulsions disappeared for about two months, but the tumor grew through the opening of the skull, attained considerable size on the outside of the skull, and the symptoms returned. Wiener speaks in favor of trephining in cases of brain tumor, even when there is no other hope of success than the relief of the general symptoms.

Rohmer[2] has collected a large number of cases in which the palliative operation in tumor of the brain had a beneficial effect on vision.

von Bergmann[3] has called attention to the fact that in contrast to those cases in which improvement was pronounced after palliative operations for brain tumor others are recorded in which no benefit was obtained, and, indeed, the symptoms were made worse. The improvement is only temporary and in some cases has been of very short duration. The operation also is dangerous. Improvement has occurred after merely opening the skull or after opening the dura also; surgeons, therefore, he said, preferred to remove a large piece of bone and leave the dura intact, but as cerebrospinal fluid can escape only after the dura is opened, the incision of the dura is a necessary part of the palliative operation. There was a tendency, he said, to stop at a palliative operation, and this must be strongly combated. The future of the surgical treatment of brain tumors lies in the advancement of diagnosis, and in this way the almost thankless palliative operation may be changed to a radical cure. As the escape of cerebrospinal fluid is more important than the opening of the skull, v. Bergmann recommended lumbar puncture. Since v. Bergmann wrote his book experience has shown that lumbar puncture in cases of brain tumor may be dangerous. He has been pessimistic both as regards palliative and radical operations for brain tumor. He gives a long list of cases in which exploratory and palliative operations were done, but almost all of these were attempts at removal of a tumor. In his book (1899) occurs the expression, "decompressive trepanation." He refers to a case reported by Remsden in 1825, in which trephining was done for headache.

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[1] New York Med. Jour., Oct. 15, 1898, p. 541.

[2] Revue Méd. de l'Est., 1898, p. 525.

[3] "Die Chir. Behandlung von Hirnkrankheiten," Berlin, 1899.

Sänger,[1] in 1894, reported two cases to show that under certain circumstances trephining is proper, even when a tumor cannot be removed. In 1899 he presented a case of brain tumor in which a palliative operation had been performed. Inunctions were at first effective, but later the symptoms became very severe. An opening was made over the cerebellum and the cerebellar lobe at once protruded; when the dura was incised much cerebrospinal fluid escaped into the bandages for more than three weeks. After the operation vomiting and headache ceased and full recovery seemed possible. Choked disk disappeared, and vision improved so much that the patient was again able to read. The gait also improved and ataxia became hardly noticeable. The operation was performed August 7, 1899, and the presentation of the patient was made November 14, 1899.

Babinski,[2] in 1901, reported a case in which a tumor could not be found after the cerebral dura was opened, although the symptoms had been so severe as to make an operation desirable. The headache and vomiting ceased after the operation, which was performed January 14. The choked disks disappeared by January 30. The presentation of the case was made February 7, 1901. In another case Babinski observed signs of an intracranial disease. The patient had an intermittent discharge of cerebrospinal fluid by the nose. Headache was lessened by the escape of fluid and became more intense when the flow ceased. Nature in this way produced decompression he says. This case is not unlike some others reported in the literature in which Nature has effected a means of relief for intense intracranial pressure, and bears a resemblance to a case observed by me in consultation with Dr. Laplace and reported in this paper (Case 1).

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[1] Neurolog. Centbl., Dec. 1, 1899, p. 1118.

[2] Revue Neurolog., 1901, p. 266.

A. Sänger[1] presented a case in which he had made the diagnosis of cerebellar tumor and an opening was made over the left cerebellar lobe. The improvement was marked, and Sänger believed that either the tumor had undergone a retrograde change or the condition was hydrocephalus. Of eleven cases in which a palliative operation was performed the symptoms were much lessened in ten. The time for operation that Sänger chooses is the beginning of impaired vision. When the opening is made over the cerebellum the dura must not be opened immediately.

In 30 cases of brain tumor studied by Leslie Paton[2] that were operated on, useful vision was saved in 22 and the vision was as good as before the operation in 18. It is impossible to draw conclusions regarding his cases in which a tumor was removed and those in which a palliative operation was performed. In the discussion following Paton's paper J. S. Risien Russell said he had no doubt that trephining was of great value in saving sight. He never hesitated to recommend the operation even in cases in which there was no chance of either localizing the tumor or removing it, for by relieving the intracranial pressure alone sight could be saved. His experience had been that removal of bone alone was not sufficient; it was usually necessary to open the dura in order to obtain sufficient relief of pressure to bring about a subsidence of the optic neuritis. Paton also expressed himself in favor of opening the dura, as he did not believe merely opening the skull was of much benefit. In two cases only the cranial cavity was opened, and blindness developed in both.

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[1] Verhandl. d. Deuts. Gesell. f. Chir., 1902, p. 158.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1904–1905, vol. xxv, p. 129.

Codman[1] says:

> "In 28 of the 36 operations the attempt at a radical removal of the tumor was made, and in the eight worse cases only was it decided to do only a palliative operation. I think we may almost say it is certain that if in no case the attempt at radical removal had been made and, instead, a simple operation for the relief of pressure had been done through an intermuscular incision over a "silent" portion of the cortex, the mortality would have been greatly diminished and the percentage of improvement been made much greater. I do not mean to say by this that radical operation in the hands of a surgeon who has made a specialty of brain work is not justifiable, but that in the hands of most of us, a simple palliative operation will be far better in the long run."

In my opinion, only one who has made a specialty of brain surgery should attempt the removal of a tumor of the brain.

J. J. Putnam[2] reports cases of palliative operation. In Case 2 marked relief from terrible headache and optic neuritis followed the making of a large trephine opening in the parietal region, and this, too, although the bone was replaced. This improvement was attributed to the escape of cerebrospinal fluid, which gushed in large quantities from the wound in the parietal dura. The tumor was a glioma of the lateral lobe of the cerebellum. In Case 3 a large tumor mass protruded through the trephine opening, pushing the skin before it. A measure of relief followed the operation.

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[1] Boston Medical and Surgical Journal, July 20, 1905, p. 74.

[2] Boston Med. and Surg. Jour., July 20, 1905, p. 76; also Jour. of Nerv. and Ment. Dis., May, 1906

Recently, in association with Watermann, he has reported other cases. Lund[1] also has reported improvement from the palliative operation in cases of tumor of the brain.

Cushing[2] has reported recently cases in which the palliative operation has been of much benefit.

I add condensed notes of two unreported cases in which palliative operation was performed by Dr. W. W. Keen with improvement in each.

Case 1.—Jan. 3, 1901, E. H. M., aged twenty-six years, complained of failing vision and had later several attacks of temporary blindness. Among the first symptoms were severe headache on the right side and vomiting, later the pain shifted to the left side. He was found to have extensive optic neuritis. He has a little vision now in the left eye but is blind in the right eye. He has sharp, boring pains which shoot from the upper occipital region to the back of the left ear. The patellar reflexes are exaggerated. Dr. de Schweinitz, Dec. 17, 1900, found optic neuritis passing into atrophy.

Dr. Keen, Jan. 30, 1901, made an osteoplastic flap over the left parieto-occipital region. The dura was very tense. Four small openings were made in the dura, but nothing abnormal was discovered. The brain was punctured in the attempt to reach the lateral ventricle, and the attempt probably was not successful. The piece of bone was removed entirely. The patient was discharged February 18, almost entirely relieved of his headache.

At Dr. Keen's suggestion, I made inquiries regarding the present condition of this man and received the following letter, dated April 20, 1906: "I am wonderfully improved and relieved as to pain, cannot see except to distinguish between night and day, have not been bedfast for a year or more, at intervals have severe pain, say every week or two, lasting from twelve to twenty-four hours, after which brain is very sore. Since last July have been having a discharge from the left nostril of a watery-like fluid, since then the pain hsa not been quite so severe."

Case 2.—Feb. 5, 1903. E. B., aged thirty-seven years. During the summer of 1894 a small tumor developed in the scalp on the posterior portion of the right parietal bone, producing headache and a sensation of pressure. In the summer of 1895 this mass was removed. His physician stated that it was a fatty tumor. All the pain and pressure symptoms disappeared and he was perfectly well.

After several months the headache and pressure sensation gradually returned; simultaneously with the onset of the headache his eyesight began to fail, and for the last two months he has been partly blind. He has had frequent vomiting, but he attributes this to the iodides. Ataxia was noticed in the finger-to-nose test. His gait is ataxic with a tendency to fall to the right. Lateral movements of the eyeballs are ataxic. Patellar reflexes are minus. Dr. Sweet found papillitis passing into optic atrophy in both eyes.

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[1] Boston Med. and Surg. Jour., July 20, 1905, p. 81.

[2] Surgery, Gynecology and Obstetrics, 1905, vol. i, p. 297.

Feb. 11, 1903, Dr. Keen exposed the cerebellum over nearly one-half of the right half of the occipital bone. The dura was extremely tense on palpation. The patient was discharged ten days after the operation entirely relieved of his headache and with the wound healed.

Dr. John T. Howell wrote to me, April 23, 1906, that the patient died in a hospital in England, July, 1904, after an operation in February of the same year. The skull was opened above the ear and a large tumor was taken from the brain. After returning from the Jefferson Hospital, in 1903, the patient's improvement was of short duration.

WHEN PALLIATIVE OPERATIONS ARE ADVISABLE.

After a careful study of these cases and of the views of the different authors, it will be seen that the weight of opinion is decidedly in favor of palliative operations. The choked disk, headache, vertigo, nausea, vomiting, and, to some extent, the convulsions are all favorably influenced by this method of treatment. Relief from these distressing symptoms is by no means to be despised, even though the tumor is not removable. The relief from many of these symptoms is often permanent, *i. e.*, during the period the patient may continue to live, and, as the growth of the tumor is not hastened by the palliative operation and may be slow, we are thankful for a means of removing the distressing symptoms of intense intracranial pressure.

My first case of palliative operation was in 1898, and the operation was performed by Dr. John B. Roberts. The marked improvement that occurred in that case and persisted at least during several years and until the patient passed from our observation convinced me of the benefit to be derived from palliative operations (Case 2 of our series). She is still living and is in good health.

In one of our cases (Case 10) headache was so severe that relief was demanded at once. Byrom Bramwell, as already mentioned, has attributed sudden death in some cases of brain tumor to the severity of the headache and sudden inhibition of the action of the heart. The patient to whom I refer suffered such intense pain that he could not be reasoned with and was not responsible for his acts. Occasionally, in his attacks of pain, he grabbed anyone who might come within his reach and once bit his mother through the arm. The relief afforded in this case by a palliative operation has been most striking. Indeed, his relief from the previously constant pain has been so great that, as he expresses it, he is "lonesome without his headache."

In another case (Case 11) headache has been the symptom that above all others demanded treatment and the pain in the head was associated with pain in the neck, shooting down into the upper limbs. The symptom-complex has been in large degree that of cerebellar tumor, but the pain shooting into the upper limbs has made me fear that the tumor was extending downward through the foramen magnum on the cord. Some years ago I had a case of this kind, and at the necropsy a flat tumor was found covering a part of the pons extending into the posterior part of the fourth ventricle and growing down on the cord for several inches. In that case the development of the symptoms in the upper and lower limbs was more rapid than in the present instance, and, therefore, I feel that the tumor in the latter case may be confined to the region of the cerebellum. If so, I have no explanation for the pain in the upper limbs. The decrease of this pain, together with the decrease of the headache after the dura was opened at the second operation, gives me reason to believe that probably this pain of the limbs was indirectly caused by the cerebellar tumor. Headache and pain in the upper limbs still occur occasionally, but the condition of the patient has been vastly improved by the removal of a piece of bone and by opening the dura over the cerebellum.

I am somewhat skeptical as regards disappearance of Jacksonian convulsions after merely opening the skull and dura if the convulsions are very frequent.

In one of my cases in which the brain was exposed by Dr. Edward Martin and which will be reported by Dr. Martin and myself later, the tumor could not be found at the time of operation. The man was having five or six Jacksonian convulsions within an hour, and relief of intracranial pressure did not in any way check them. He died from exhaustion following the numerous convulsive seizures on the second day after the operation. After death a minute tumor was found nearly in the centre of the area exposed, but a little subcortical.

In a still more recent case under my care, in which fracture of the skull and subdural hemorrhage had occurred, trephining probably prolonged life about twenty-four hours, but did not in any way check the Jacksonian convulsions which before the operation were as frequent as six or seven within an hour, and continued in weakened intensity but with equal frequency after the operation. Opening the skull does not seem to relieve acute irritation of the brain, but it is of benefit in chronic cases, as not rarely convulsions occurring infrequently have been rendered less severe by this procedure.

I have considered whether palliative operations can obscure focal symptoms. Bruns thinks they cannot, but the subject is one that has important bearings. It is probable that a small tumor will cause more local disturbance if the general intracranial pressure is increased, not only because the local disturbance is added to the general pressure, but also because any one part of the brain is more irritable when the disturbance of the brain is general. This, however, is not sufficiently important to prevent palliative operation, and I would advise that this should be done before the general symptoms become very intense, and especially before optic neuritis has developed so far that blindness is likely to result. The apparent unanimity of opinion as regards the effect on choked disks of opening the skull makes the necessity of this operation at an early period very evident.

It is a mistake to regard palliative operations as a substitute for radical operations. The tumor should be removed whenever this is possible, and palliative measures are to be considered only when the tumor cannot be located, or is too large for removal, or possibly is a glioma. Palliative operations only relieve, except in extraordinary cases such as those reported by Horsley. Codman's views that palliative operations are preferable to radical operations I cannot share, and would add that the argument he offers is insufficient, viz., that the former are less dangerous in the hands of inexperienced surgeons. No surgeon who has had little experience in operating on the brain should attempt the removal of a brain tumor, and before advising the attempted removal of a brain tumor I always inquire who is to perform the operation.

The experience of Horsley as regards atrophy of tumors as a result of palliative operations seems to be unique; at least I have found no other similar cases, nor have I ever seen any such result in any of our cases. Broca and Maubrac doubted whether Horsley was correct in believing that partial ablation of a tumor may retard the growth of the rest, but Horsley has observed atrophy of a tumor following a simple opening of the skull and dura. This is a striking statement. I have never seen arrest in the growth of a tumor following the removal of a part, but several times I have seen increase of symptoms result, and I dread the partial removal of a growth, especially if it is a glioma. It has seemed to me that the congestion of the tumor and of the surrounding tissue and the greater space afforded for the growth of the tumor, after partial removal, favor the rapid growth of the remaining portion. I am almost convinced that it is better to leave a tumor untouched if only a part can be removed, especially if the growth is a glioma. The attempt, I think, should never be made to remove a glioma, and yet there seems to be no way to determine before operation that the tumor is a glioma. A patient has been under our care with the diagnosis of brain tumor and was operated on several times by Dr. Frazier. Each time as much of the tumor as could be seen was removed, and yet a return of the symptoms occurred soon after each operation, and when a necropsy was obtained the tumor (a glioma) was found to extend from the upper to the lower surface of one cerebral hemisphere.

I doubt whether it can be held that a palliative operation, by which the tumor is not partially removed, by relieving pressure, favors the growth of an intracranial tumor. Unquestionably the brain has protruded through the opening as in the cases of Schlesinger, Frazier, and Cushing, but it seems a questionable proceeding to excise the protruding portion. The tumor has grown through the opening and on the outside of the skull (Jolly, Wiener, Putnam, and others). In Jolly's case it formed an extracranial mass nearly as large as the man's head. It seems unquestionable that life was prolonged in this case by the operation, although it is doubtful if life under such circumstances is worth having. In Jolly's[1] case the clinical signs extended over a period of thirteen years. In the case that Dr. Martin and I observed, to which reference has been made, the symptoms of brain tumor had existed for eight years.

We have had some extraordinary cases in which the symptoms of brain tumor disappeared entirely or almost entirely after an opening had been made in the skull and dura; such, for example, is the case observed by Dr. John B. Roberts and myself in 1898, and also Cases 4, 5, 6, 9, 10, of our series. It may be thought that the growth of the tumor is arrested by the operation; this is possible, but doubtful. Internal hydrocephalus (meningitis serosa) or some other lesion may give the symptoms of brain tumor, and relief of intracranial pressure may cause great modification or disappearance of the symptoms. In some cases in which relief has followed palliative operations probably an incorrect diagnosis has been made; in others, the relief of pressure has permitted the tumor to grow slowly without causing many symptoms. My views as a result of my experience and of a study of the literature are:

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[1] Berl. klin. Wochft., July 17, 1899, p. 636.

1. Palliative operations should be performed early in every case in which symptoms of brain tumor are pronounced and before optic neuritis has advanced far, especially when syphilis is improbable or antisyphilitic treatment has been employed.
2. Partial removal of a tumor, especially of a glioma, is a questionable procedure.
3. Palliative operation does not under ordinary circumstances cause atrophy of a brain tumor and probably does not arrest its growth; on the other hand, it probably does not hasten its growth.
4. Palliative operation is not to take the place of a radical operation when the latter can be performed without great risk to the patient.
5. In some cases the symptoms of brain tumor disappear almost entirely for a long time or permanently after a palliative operation. This result is obtained either by relief of intracranial pressure or by removal of some lesion (meningitis serosa, etc.) other than brain tumor, and yet causing the symptoms of tumor.

REMARKS BY DR. FRAZIER.

The question as to whether a palliative or radical operation is indicated may be determined by answering the questio ans to whether the tumor is operable or not. The operability of brain tumors has frequently been discussed and the facts and figures which have been presented to us have been based almost exclusively on the autopsy records and not on the finding at the time the operation was performed. The autopsy records may help to determine the percentage of operable tumors in so far as it reveals the nature of the tumor and may be able to fix on its point of origin. The very vascular infiltrating sarcomata should be classed with the inoperable tumors, for I believe the expectation of life and the enjoyment of that much of life which remains to the patient would be greater in the majority of cases after a palliative operation. On the other hand, the fibroma, or the somewhat defined fibrosarcoma and the gumma of limited dimensions, should be classified as of the operable group. The size of the tumor as revealed at the autopsy has no practical bearing at all on the problem; the size of the tumor at the time the symptoms first made a diagnosis possible should only be considered. There are some tumors of such large proportion as to make any attempt at removal inadvisable, but, on the other hand, tumors of very small dimensions may lead to a fatal termination.

If it were possible to ascertain the point from which the tumor took its origin we should have some valuable statistics as to the operability of tumors. The further away the tumor is from the cortex or the nearer it is to the base of the brain the more it approaches the field of inoperable growth If the statistics are to be of much value to the surgeon they should be based on the records which are made on the operating table or at the autopsy, if the patient dies as a result of operation, only in those cases in which the patient has been brought to the surgeon comparatively early in the course of the disease. One might as well prepare a table and compile statistics as to the operability of carcinoma of the stomach based on the autopsy records of patients who died in the most advanced stages of the disease, when the lymphatic nodes far beyond the primary lesion have been invaded and the tumor is no longer confined to the stomach, but has invaded other organs adjacent to it. The question as to what percentage of tumors of the stomach may be removed must be and is being determined in the very earliest stages and on the operating table. So with tumors of the brain, the greater the number of cases brought to the surgeon in the earliest recognizable stage of the disease the sooner will we be able to say with some degree of accuracy in what percentage of cases there is a reasonable hope of being able to perform a radical operation.

There are two classes of cases in which a decompressive operation may be required. One in which there is reason to believe the tumor cannot be removed in its entirety and the other in which the tumor cannot be localized and yet the possible loss of vision, the intense headache and distressing vomiting almost demand the immediate adoption of some measure of relief.

Cases of tumors of the cerebrum in which localization is impossible are rather the exception, although in cerebellar lesions it not infrequently happens that the tumor cannot be localized.

A word or two might be said with reference to operations performed for the relief of cerebellar tumors. The anatomic relations of the structure in the suboccipital resemble in certain particulars those in the temporal region. In both the bone is covered with a well-developed musculo-aponeurotic layer, even of a greater bulk in the suboccipital than in the temporal region. For purposes of exploration it is necessary to reflect a flap of considerable size, the incision beginning at the mastoid process and following the line of the transverse sinus to the occipital protuberance, drops in a vertical direction along the median line. And when the flap is replaced great pains should be taken to approximate carefully not only the cutaneous, but the musculo-aponeurotic layer. When the tumor cannot be localized or for any other reason a radical operation is not deemed feasible a less mutilating incision will answer the purpose quite as well. A vertical incision should be made, beginning a little above the superior curve line and extending downward for a distance of three or four inches. The edges of the wound can be retracted sufficiently to afford space necessary to carry on the subsequent steps of the operation. A much more perfect approximation of the wound may be obtained as the muscles are split in the direction of their fibres and not in various angles, as is the case when a curved incision is made following the line of the sinus. This vertical incision enables one to remove the bone only from one side, but if for any reason a bilateral craniectomy seems advisable, the muscle-splitting operation may be repeated on the opposite side. Such a procedure has many advantages over the conventional bilateral craniectomy in which the bone from one mastoid process to the other is removed. There is less chance of disturbance arising from traction on the pons or medulla if an intervening bridge of bone is left. In addition to this the bone in the region of the occipital region is very vascular, often riddled with vascular channels of such dimensions as to admit of a very profuse and sometimes alarming hemorrhage. The region immediately surrounding the occipital protuberance should be regarded as the dangerous zone and as such scrupulously avoided.

Failing to find the tumor, we have in certain cerebellar cases deliberately removed from one-quarter to one-third of the cerebellar hemisphere and have attributed the relief of pressure, in part at least, to this. In all our cases the operation has been confined to one side, and as yet we have not found it necessary to operate a second time for the removal of the bone on the opposite side. Even if it were conceded to be necessary to remove eventually the bone overlying both hemispheres the operation had better be performed in two stages rather than in one; patients with cerebellar lesions are at the best not very favorable subjects for operative intervention, and preference should be given, therefore, to this precedure, which in giving the desired results will require less time and be attended with less hemorrhage and traumatism of the tissues.

First, as to those cases in which the tumor cannot be localzed. There are two points in the technique of the operation about which there may be some difference of opinion, namely, the area of the brain to be uncovered and the incision of the dura. The decompressive operation consists solely in the removal of a portion of the cranial vault with or without incision into or removal of a portion of the dura. When there is no guide as to the situation of the tumor the operator is free to select either one of four areas: the frontal, parietal, occipital or temporal. In discussing this question several years ago, attention was called to the desirability for cosmetic reasons of avoiding both the frontal and occipital regions; there remain, therefore, but two areas from which to make the election: the parietal and the temporal. It is desirable, as Sänger suggests, to give preference to that portion of the skull beneath which may be said to exist a silent area of the brain. This is especially true of the right temporal region, so that, in the absence of any contraindication, this may be said to be the region of choice. Through a longitudinal incision, following the direction of the fibres of the temporal muscle, a section of bone equivalent to 6 or 8 square centimetres may be removed. One should be content to make but one opening, at least at the first sitting. This will be found in most cases to be sufficient to afford the necessary relief of pressure. If in the course of events there is evidence of a recurrence of increased tension, as shown by such symptoms as headache, vomiting or choked disk, a second opening may be made on the opposite side of the head. Such were the circumstances in one of our patients (Case 5). The headache, which had been relieved temporarily, recurred. A second opening was made on the opposite side of the head, and since that time, a period of about three years, the patient has enjoyed almost entire relief.

Speaking now only of those cases in which the tumor has been definitely localized, it is impossible to determine prior to the operation whether the tumor is one appropriate for a radical rather than a palliative operation. The duration of the disease is a guide neither to the size nor to the nature of the tumor. Malignant tumors in the brain are many of them surprisingly slow in their growth, and, though of many months' or years' duration, may still be in the operable stage. A very large tumor may be present with comparatively slight disturbance, or a very small one may be very serious in its consequences. A tumor of several years' duration may be no larger than a hickery nut or walnut, and yet give rise to a condition which may ultimately prove fatal.

Recently I was called by Dr. Charles K. Mills to operate on a patient who had had symptoms of a tumor for more than a year. The localizing symptoms were very vague, but all seemed to point to a lesion of the left cerebellar hemisphere. This was thoroughly explored, but with negative results, except in so far that there seemed to be a very unusual degree of tension within the cerebellar fossa. This was so suggestive of the presence of a tumor as to justify a very careful search. The results were negative. About two weeks later the patient died, and a tumor as large as a peach was found compressing the under part of the occipital lobe directly in contact with the tentorium cerebelli. Its position satisfactorily explained the existence of cerebellar symptoms. As the brain was being lifted out of the cranium at the autopsy the tumor dropped on the table. Though of large dimensions it was clearly an operable one, and had the symptoms made it possible to localize the growth it could have been removed without any difficulty, and owing to its benign nature the operation would have resulted in a complete cure. In such a case had the operator proceeded deliberately to the performance of a decompressive operation and not attempted to explore the affected area the opportunity of saving the patient's life and of affording him permanent relief would have been thrown away.

In every instance, therefore, the operator should begin the operation with the intention, first, of attempting to expose the tumor. He should proceed as for the performance of an osteoplastic operation; the flap should be carefully mapped out and reflected and the usual exploratory measures adopted. Failing to find the growth or finding an inoperable one, the propriety of a decompressive operation is clearly indicated. He must then determine as to whether he will remove the bone of the flap, wherever this may be, or close the flap and remove the bone from the area of choice, namely, over the right temporal lobe. When, as is true in the majority of cases, the tumor is situated in the postfrontal (motor) or parietal region the bone over the temporal region may be removed through the same opening, thus avoiding the necessity of another incision. This was the procedure adopted in one of our reported cases. The patient was suffering so intensely from headache that he was operated on the day of his admission to the hospital; not waiting to make a more careful study of the case with a view toward accurately localizing the growth, we proceeded at once with the intention of performing a decompressive operation. Sufficient was known of the case to lead us to believe the tumor was in the motor region. Accordingly this was laid bare and a very large vascular tumor, probably a sarcoma, was at once exposed. Deeming it to be inoperable and wishing to afford relief from the intracranial tension, only the lower half of the bone contained in the flap, which extended down to the Sylvian fissure, was removed, but through the same opening a considerable portion of the bone lying below the fissure and under the temporal muscle was also removed with rongeur forceps. As will be seen in the record (Case 10) the headache was immediately relieved. The most important question, however, in the technique of the decompressive operation is as to whether the dura shall be incised or removed. The region from which the bone is to be removed is by comparison a matter of slight importance. To be able to answer this question one must be in a position to state from experience, first, whether the escape of cerebrospinal fluid through a dural opening is essential, and secondly, whether sufficient relief of tension may be afforded with the dura intact. As to the escape of cerebrospinal fluid there are certain observers, notably v. Bergmann, who believe that the beneficial effect of this operation is largely attributable to the escape of cerebrospinal fluid. It might be difficult, possibly, to prove or disprove this idea on theoretical grounds, but from our own experience we have not been able to note any radical difference in the results between those cases in which the dura was either left intact or if incised for exploration was closed immediately afterward. To the escape of a small quantity of cerebrospinal fluid through a temporary opening in the dura could scarcely be attributed the relief, which extends over a period of months or years. In so far, therefore, as the results of the operation are concerned, it makes no difference whether the opportunity for drainage was afforded by a permanent opening in the dura. In the most satisfactory of the cases under our care the dura was closed; in one case, particularly, in which an exploratory dural incision was not entirely closed, the patient enjoyed no appreciable benefits from the operation.

I think it is very generally believed by those who have had but little experience with these operations that the success of the operation depends on the establishment of an opening in the dura as well as in the skull. It is said that the dura is not elastic enough to stretch sufficiently when subjected to pressure. That the dura will stretch, when the overlying bone is removed, sufficiently to afford adequate relief of tension has been demonstrated in our cases over and over again. Take, for example, a case which was operated on for a tumor believed to be in the occipital region. The tumor was not to be seen on or near the cortex, but there was a very much dilated right ventricle, possibly due to a tumor at the base, cutting off the intraventricular communication. If we turn to the illustration (Fig. 1) we see a genuine hernia cerebri, a hernia in which the dura as well as the scalp form the coverings. This patient has been under observation for a number of years and for the past year the hernia has not increased in size. The optic neuritis was in this case too far advanced to enable us to save the patient's vision: in other respects the patient enjoys excellent health and is now in attendance at an educational institution. A decompressive operation was

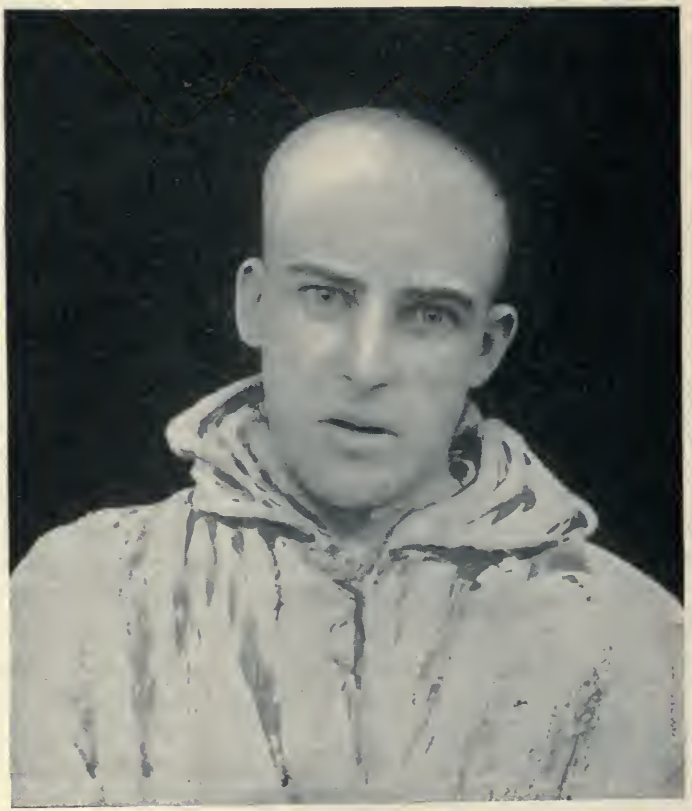

FIG. 1.—Showing the protrusion subsequent to the development of a hernia cerebri following a decompressive operation, in which, after exploration, the dural wound was closed.

not a matter of choice in this case; at least at the outset this was not contemplated. The brain bulged so through the opening that it was impossible to close the flap without removing the bone.

As an example of how rapidly the dura may stretch when subjected to excessive pressure, one of the cases of our series may be cited in which an exploratory craniotomy revealed a large and very vascular ill-defined sarcoma. Its removal was out of the question; the incision in the dura was closed and a section of bone removed. Several records were made of the convexity of the head (Fig. 2) at short intervals, and

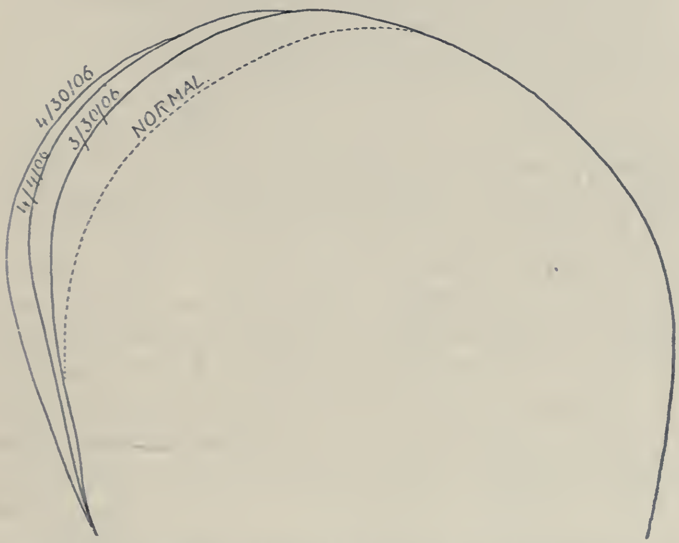

Fig. 2.—Showing the rapid development of a hernia cerebri in the decompressive operation. After the operation the dural wound was closed. The dotted line represents the normal contour on the affected side. The other lines represent the contour of the head: (1) March 30, 1906, one week after the operation. (2) April 4, 1906, twelve days after the operation. (3) April 30, 1906, five weeks after the operation.

it is rather surprising to find how rapidly the dura had expanded to accommodate the growth. On the other hand in another case in which a tumor was exposed and found inoperable the dural incision was only partially closed. The results in this case were as compared with the other, in so far as the relief of symptoms was concerned, much less satisfactory.

Whatever else may be said either in favor of or against the removal of the dura it should be borne in mind that if the dura is removed and any complication arises in the repair of the wound it is more than likely that a fungus cerebri will develop. An accidental wound infection, the persistence of a sinus, the remains of a drainage tract may result in infection of the exposed brain tissue and that very fatal condition a fungus cerebri. In the hands of operators experienced in this field of surgery the danger is very slight, but it is questionable whether the removal of the dura should be resorted to as a universal practice. While in a given number of cases the results may not be so pronounced the mortality will be higher unquestionably if the dura is removed in every case. Inasmuch as the brain may herniate through the opening in the skull when the dura is left intact, there is really no necessity for removing more than the overlying bone. If the dura is disturbed at all, the operator should be content with making a crucial or radiating incision in it, and in many cases it may be advisable to reserve this step of the operation until indications arise for further intervention. This course was pursued in one of our cerebellar cases in which temporary relief having been afforded the symptoms, particularly pain, recurred.

A muscle-splitting incision was made, the edges of the wound retracted and a crucial incision made in the dura. The results were very gratifying and have continued so now for a period of several months.

MORTALITY. Every one of the twelve patients operated on at the University Hospital, eleven by Dr. Frazier and one by Dr. Martin, recovered. The absence of any fatalities in the entire series is worthy of note. When one is debating as to the advisability in a given case of performing a radical or palliative operation, consideration should be given to the fact that there is at least no immediate risk as to life from the decompressive operation. In the palliative measure we have an operation which almost inevitably relieves the sources of discomfort and suffering, not only without endangering but frequently prolonging life; in the radical measure we are undertaking an operation, which has a comparatively high mortality and is only successful in a small percentage of cases in finding and removing the tumor.

Results. The opinions expressed in the paper are based on observations in a series of fourteen cases. The first operation was performed in 1898 and the last March 20, 1906. In but one of the entire series did the operation fail to afford the relief which would justify its performance. There was in this case some immediate improvement, but so slight as not to warrant the promise of relief which was held out to the physician and relatives of the patient. The operation was performed in March of this year; the patient was at the time almost comatose and her life was in immediate danger. At the present writing, May 25, 1906, her physican, Dr. Thomas O. Nock, writes me that the patient has been moved to the country, but her condition is little, if any, improved. All that can be said of this case is that life has been prolonged. The results in this case are a striking contrast to those following an operation performed in October, 1903. This patient probably has two lesions, one in the parietal lobe and one in the cerebellum, and before the operation suffered intensely from headache and neuralgia. When last heard from two and a half years after the operation she still enjoyed absolute relief. Perhaps the most brilliant of the series was a patient who presented the clinical symptoms of a cerebellar lesion, intense choked disk, frightful headaches, and persistent nausea, vomiting and ataxia. This patient (Case 4) was operated on two and one-half years ago and when last seen, February of the present year he was the picture of health and was engaged in hard manual labor. Neither headache nor nausea recurred, and though he had a choked disk, so elevated that he could scarcely recognize large objects, his vision is now practically normal. Apart from the fact that he occasionally has attacks of what he calls "dizzy spells" he is to all intents perfectly well and now weighs 184 pounds.

The decompressive operation offers to the patient relief from the three cardinal symptoms of cerebral tumor, headache, nausea, and vomiting, restores vision, and if the lesion is in the cerebellar fossa relieves ataxia and vertigo. Mention has already been made of the dangers of postponing operation once there are signs of choked disk. One of the strongest arguments in favor of the palliative operation is the opportunity to save or restore the patient's eyesight. Without exception the choked disks subsided in every instance but unfortunately in at least three of our cases the patients remained totally and hopelessly blind because the optic neuritis was of so long duration that the nerve had undergone atrophy. The subsidence of the choked disk takes place as rapidly when the dura is left intact as when it was incised. The case, which in Dr. de Schweinitz's opinion and experience responded most promptly to the operation was one in which the dura was not disturbed. Although the operation was performed only seven months ago the patient's vision has continued to improve. The prospects of preserving the eyesight alone would justify the operation, but no less so the opportunity of relieving headache. Our results with respect to headache have been uniformly good. There were two or three cases in which the headaches did not entirely cease, but in these there were intervals of relief and the headaches were less severe. As to the duration of the period of relief the longest in our series is between two and one-half and three years. In at least three of our cases the intensity of the pain seemed to be as great as that of a tic douloureux, and one patient had become addicted to the liberal use of morphine; yet in each one of these the relief was immediate, absolute, and permanent. In one instance the suffering was so intense that the operation was performed within a few hours of the patient's admission to the hospital, before an opportunity was afforded to determine with any degree of accuracy the location of the tumor. What has been said of the beneficial effect of the operation on the choked disk, even when the dura was left intact, may be repeated in dealing with headache. Not that the dura was not opened in some cases for exploration, but in almost every instance the dural wound was immediately closed with sutures.

Mention might be made of the fact that in two instances Nature had already begun to relieve the intracranial tension. In these cases the tumor had grown through the dura and in one instance had almost perforated the skull. In conclusion we may say that there is no form of treatment, either medical or surgical, which, in the presence of an incurable lesion, causing sometimes great discomfort, sometimes intense suffering, so uniformly affords so great and enduring a measure of relief.

Summary of Cases.[1]

	Location of lesion.	Period since operation.	Results.
1. L.	Cerebrum	1 year (operation was not performed but relief of pressure occurred spontaneously).	Alive and free from pain.
2. B.	Cerebellum	7 years.	Almost complete recovery during two or three years; present condition unknown.
3. G.	Cerebellum and cerebrum	2 years and 7 months.	Alive and free from pain.
4. E.	Cerebellum	2½ years.	Alive and free from pain.
5. D.	Cerebellum	2½ years.	Alive and free from pain.
6. B.	Cerebrum	1 year and 7 months.	Alive and free from pain.
7. M.	Cerebellum	8 months.	Second operation was performed; tumor found and removed.
8. K.	Cerebellum	7 months.	Died seven months after operation.
9. L.	Cerebellum	7 months.	Alive and free from pain.
10. S.	Cerebrum	2½ months.	Alive and free from pain.
11. F.	Cerebellum	2½ months.	Alive and free from pain.
12. D.	Cerebellum	2 months.	Radical operation was performed two months after palliative operation.
13. C.	Cerebrum	....	Alive but little if any relieved.
14. S.	Cerebellum	About 3 years.	Improved; death from tumor about three years after palliative operation.

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[1] A case under the care of Drs. Spiller and Martin to which reference is made in this paper, is not included in this table.

ORIGINAL OBSERVATIONS.

Case 1.—Probably tumor of motor area. The following case is of great interest as it seems to be one in which relief of symptoms from intracranial pressure occurred by escape of cerebrospinal fluid through the cribriform plate of the ethmoid bone without operation, and it may be, therefore, regarded as a case in which relief was afforded in a manner similar to that in palliative operations.

Examination. L. was seen by Dr. Spiller in consultation with Dr. Ernest Laplace, Aug. 13, 1904, at which time the following notes were taken: In showing the teeth the left corner of the mouth is drawn up better than the right. When drawing up each corner of the mouth separately he draws up the left better than the right. The eyelids are closed equally well on the two sides, and the resistance to any attempt to passively open the lids is as good on one side as on the other. Percussion of the scalp shows no area of tenderness. When the tongue is protruded it tends to deviate a very little toward the right. The movements of the tongue are free in all directions, and the tongue is not atrophied. The movements of the eyeballs seem to be normal in all directions, except that the movement to the left may be a trifle impaired. There is no nystagmus. The reaction of the iris to light is preserved, but tardy on both sides. Convergence and accommodation cannot be tested satisfactorily on account of the mental condition. The pupils are equal and regular. The masseter muscle contracts well on each side, but the left masseter contracts a little better than the right when the patient bites. He says that he feels touch more distinctly on the left side of his face. Pin-prick is probably perceived in the right side of the face as well as in the left, but his statements regarding sensation are not to be relied on. He understands what is said to him, although he appears dazed and has difficulty in replying, but apparently is not aphasic.

Upper limbs: The grasp of the right hand is distinctly impaired and is decidedly weaker than that of the left hand. The upper limbs are well developed and equally so. The biceps and triceps tendon reflexes on the right side are distinctly prompter than the corresponding reflexes on the left side. After a spasm the right upper and right lower limbs seemed to be almost completely paralyzed, and Dr. Laplace says that this paralysis following a spasm is only temporary.

Lower limbs: The lower limbs are well developed and there is no atrophy in any part. The patellar reflex is about normal on the left side, but exaggerated on the right side. There is no ankle clonus on either side. The Achilles tendon reflex is about normal on each side, and a trifle prompter on the right than on the left. The Babinski reflex is very pronounced on the right side; the big toe as well as the adjoining toe being drawn upward distinctly. The Babinski reflex is not present on the left side, the movement being flexion.

During the examination an epileptic attack occurred. It began with a slight flexion of the right forearm on the arm. This movement lasted from thirty to sixty seconds before the face was involved. The right lower limb seemed to be involved before the face. Clonic moved ments of small extension began in the right lower limb, afterward the movements occurred in the right upper limb. Both right upper and lower limbs were very spastic. The right side of the face was thrown into violent tonic spasm, and the lids on both sides were equally affected. There seemed to be a slight tendency of the eyeballs to turn to the right, but this was not pronounced. The lower part of the left side of the face, the left upper and lower limbs showed no convulsive movements and the left limbs were not spastic. Consciousness did not seem to be lost during the attack. Urine was not voided during the attack, but was immediately after the attack, voluntarily. After the attack the man was drowsy and passed into a light sleep lasting about five minutes.

Further Examination. A further examination on August 14th gave the following results: Sensation for touch is more acute on the left side of the face; sensation for pain is more acute on the right side of the face; this agrees with the findings of yesterday. The grasp of the right hand is not so good as it was at the beginning of the examination yesterday. A knife or a pencil put into the right hand is distinguished without difficulty when the eyes are closed. The weakness of the right upper limb has been much greater since the attack that we saw yesterday than at any time previously. He can barely lift the right hand from the bed. The paralysis of the right lower limb which developed after the attack that we saw yesterday disappeared in part during the afternoon, whereas, that of the right upper limb has persisted with very little change. The sensation for touch and pain seems to be more acute in the right lower limb than in the left. He seems to understand what is said to him, but has difficulty in getting words to reply and is partly motor aphasic. He yawns repeatedly and Dr. Borsch has noticed that during the convulsive attack the eyeballs are drawn toward the right but remain in this position only during a short part of the attack. The left pupil is larger than the right and the left recovers more slowly than the right.

Subsequent History. The following notes were obtained from Dr. Laplace: The patient continued to improve under high doses of iodide of sodium (100 grains daily) until all symptoms of paralysis had disappeared. Headaches also disappeared, but the optic neuritis continued. The field of vision gradually lessened until total blindness ensued, January, 1905. The patient remained in this condition until May, when convulsions attended by severe headaches returned, exactly similar to those seen by Dr. Spiller in August. About forty convulsions took place in the course of the first week in May, 1905 He suffered with intense headaches and was suddenly greatly relieved of all headache and of nearly all the other symptoms on expectorating what seemed to be a certain amount of purulent material. (Dr. Laplace did not see the patient at the time of this nasal discharge.) Whether the material contained cerebrospinal fluid or whether it was pus which found its way into the nose, after burrowing its way through the cribriform plate of the ethmoid can, therefore, only be conjectured. In June and July the patient improved, but his lower extremities remained weak. This weakness of the lower extremities has increased gradually from July, 1905, until the present time. Mental hebetude gradually set in, but there has been an absolute freedom from headache, from July, 1905, until now.

Present Condition. Complete blindness; perfect consciousness. He is thoroughly rational when aroused; otherwise quite listless. There is great weakness of lower extremities, requiring support to enable him to walk. Both arms are quite strong. Excellent appearance; good appetite and digestion; full control of bowels and bladder. Freedom from headache. Last report says that general weakness is gradually becoming more marked. Mercury and iodides have been continued. The patient and his family have opposed any surgical interference.

Case 2.—Tumor of cerebellum (?). Mrs. M.B., was admitted to the Polyclinic Hospital, Dec. 31, 1898, under the care of Dr. Spille, and Dr. Roberts.

History. Patient had been married twenty-six years, a widow. eighteen years; had two children, twenty-five and twenty years olds respectively, at time of death. Emigrated to America five years ago, She had good health until previous to present trouble. Her present condition began two and one-half years ago with violent headacher in the left occipital region. Her symptoms have gradually increased in number and intensity, until at present they consist of very severe headache in the left occipital region, great tenderness over the same region, extreme vertigo, especially when lying on the left side in bed or attempting to walk; very ataxic gait so that she staggers like a drunken person and falls to the left side or backward.

Examination. The patellar reflexes are absent; sensation in the legs is obtunded and movements of the lower extremities somewhat restricted (she cannot cross her legs). During the last few months she has been getting rapidly worse. A diagnosis of cerebellar tumor was made, and the patient was admitted to the wards of the Polyclinic Hospital for an exploratory operation. Urinary examination: color, amber; cloudy, reaction acid; specific gravity, 1020; no albumin or sugar.

Operation. On June 4, 1899, under ether, the skull was opened by trephine and lion-jawed forceps, a hole about one and one-half inches in diameter being made over the probable seat of the lesion as indicated above. The dura mater appeared normal, as did the bone removed; the dura mater was laid back, the brain exposed, the finger inserted and the surface of the brain palpated for an area of five inches or more in diameter, but no abnormality was detected. A puncture was then made into the convolutions of the brain in several directions with a grooved director to the depth of three-fourths inch without finding any lesion. The dura mater was then sutured, and the skin flap brought into place and secured. The patient made a good recovery from the anæsthetic, but her temperature began to rise rapidly that night, reaching 101° F. at 7 P.M. Infection being feared, the dressing was removed, the sutures partially withdrawn, and the wound flushed with normal salt solution. The wound was re-dressed at frequent intervals since, all the sutures removed.

Results. June 30th. The patient has been slowly improving since the 20th, the temperature having been practically normal. The parietal region on the operated side has almost entirely lost its peculiar tenderness to touch, and the headache occurs only occasionally, is slight and is now located in the frontal region. The patient made a complete surgical recovery, and almost all her symptoms disappeared. After two or three years she returned to Europe. She has been seen recently and is in good health.

CASE 3.—Tumor of the pons. Mrs. G., aged forty-five years, was admitted to the University Hospital under the care of Dr. Charles K. Mills, October, 1903.

History. Tumor of the pons, probably second lesion in the parietal lobe; cyst evacuated; permanent disappearance of severe headache and facial neuralgia. This patient, a woman about forty-five years old, first came under the care of Dr. Mills in November, 1902. One year before coming under observation she had an attack of vertigo without unconsciousness and after this partial paralysis of the left third nerve was observed, ptosis, dilated pupil and paresis of the internal rectus being present, according to the physician then in attendance. From the history she also appeared to have had temporary paralysis of the left side of the face. She had subsequent similar attacks, some of these shortly before coming under observation. In one of the recent seizures she was found in the cellar, pacing up and down with her hands above her head, eyes dilated, and apparently struggling against suffocation. She was taken upstairs and put to bed; she tried to speak, moving her jaws without uttering a sound; her face was pale, her eyes were open. At the time of the first examinations she had paresis of the left side of the face, partial ptosis, pupils equal and responsive, the superior rectus not being as active on the left side as on the right.[1]

Examination. The patient was again examined by Dr. Mills and Dr. Spiller, Sept. 22, 1903. The left side of the face was paralyzed in the entire distribution of the seventh nerve. She complained of much pain, confined to the left side of the face and in the distribution of the fifth nerve. The masseter and temporal muscles contracted well in chewing on each side. No distinct objective disturbance of sensation on the left side of the face was made out. The left external rectus seemed to be the only external ocular muscle distinctly paretic. Astereognostic conception as tested in the right hand was almost complete.

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[1] The case is fully reported as Case 6 in a paper by Dr. Charles K. Mills, on "The Diagnosis of Tumors of the Cerebellum and Cerebellopontile Angle, with Special Reference to their Surgical Removal," New York and Phila. Med. Jour., Feb. 11 and 18, 1905.

She complained of much pain on both sides of the forehead, extending down the left side of the face to the lower border of the lower jaw. The pain did not extend below the median line of the lower jaw; it extended to the mastoid process, but not beyond this on the left side; it did not extend into the neck. She had constant pain in both temples, but it was greater in the left than in the right. All points of exit of the fifth nerve were painful to pressure; as much so on the right side as on the left. Sensation for touch and for pain on the two sides of the face seemed to be normal; she felt a touch or pin-prick as well on the left side of the face as on the right. In such instances, as when chewing a hard crust of bread, the left masseter contracted, but not so well as the right; in opening the mouth the lower jaw did not deviate distinctly. She complained of a sense of numbness in the left side of the face; this involved the entire distribution of the fifth nerve and extended on the left to the temple, but not far beyond the border of the hair. There was considerable improvement in the paralysis of the orbicularis palpebrarum. There was complete paralysis in the lower distribution of the left facial nerve. The right side of the face was normal. She did not have any nausea and seldom had spells of vertigo, although formerly the latter occurred frequently. She showed at times a tendency to fall backward or toward the left, but did not stagger as much as formerly; closing the eyelids did not affect the gait very distinctly. Hemiasynergia was not present on either side.

As the patient was slowly getting worse and was suffering extremely with pain in her head and face, it was finally decided to remove her to the University Hospital for the purpose of having an operation, which might at least be palliative. Her pain increased until it became torturing, and for days before the operation it was necessary to keep her continuously under the effects of morphine or codein. No change of any movement occurred in her local symptoms after the above recorded notes.

Operation. The operation was performed by Dr. Frazier, Oct. 14, 1903, under ether anæsthesia. A musculocutaneous flap together with the periosteum was reflected and an opening was made in the skull with the chisel and enlarged with rongeur forceps. Nothing abnormal as regards pulsation nor consistency of the left lateral lobe of the cerebellum was noted. The dural flap was reflected; at one point this flap was adherent to the underlying brain. Cerebellar tissue bulged only moderately through the wound. On exploring with the index finger in the region of the cerebellopontile angle some adhesions were separated on the lateral aspect of the cerebellum. This was followed by a gush of fluid which had evidently been walled off by adhesions. After the evacuation of the cyst the bulging subsided immediately and with a brain retractor it was possible to inspect the region of the cerebellopontile angle and to demonstrate to those present at the operation, the fifth, seventh, and eighth cranial nerves. It was noted on the blood pressure chart that when the dura was opened the blood pressure dropped thirty points and that on introducing gauze packing, or on compressing the brain with the retractor that the blood pressure rose forty points. The patient's condition was not depressed to any considerable degree by the operation; on her return to bed the pulse was 145; respirations were 40, and blood pressure was 115.

The interesting feature of the case was the ease with which the cranial nerves were exposed after the cyst had been evacuated.

The patient reacted well after the operation and passed a fairly comfortable night. There was very profuse oozing of blood and cerebrospinal fluid, necessitating frequent reinforcement of dressing. The patient was much nauseated, vomiting curds of milk and bile-stained fluid. She complained of pain in her temples. An incomplete examination showed that sensation was present on both sides of the face and no inequality of pupils was noted. On October 15th, the wound was dressed and found in good condition; the drainage was removed, there was a free flow of cerebrospinal fluid.

Subsequent History. An examination by Dr. Mills and Dr. Spiller on October 15th, resulted as follows: No cutaneous anæsthesia was present on either side; not even any hypæsthesia on the left. There was no great impairment of the motor division of the fifth nerve, the masseter and pterygoid muscles being tested. Complete paralysis in the muscles supplied by both the upper and lower branches of the seventh nerve was present. She was completely deaf in the left ear.

Slight paralysis in the right upper extremity was noticed. Astereognosis or pseudoastereognosis was present in the right hand. Anæsthesia for both touch and pain and hyperæsthesia were absent. She had had no pain in the face or head since the operation and had required no anodynes.

October 19th. The wound was again dressed and the stitches were removed; the wound was healed, with the exception of one angle; a small piece of gauze was inserted for drainage. The patient's general condition had been very good. Her pupils were normal.

24th. The surgical condition was entirely satisfactory; the wound was healed, with the exception of one spot, which was rapidly granulating. The patient's general condition had rapidly improved since last noted. She was regaining strength, had been sitting up and was quite comfortable.

29th. Patient was discharged, the surgical condition left nothing to be desired; the wound was completely healed; there was no bulging of the flap, no signs of inflammation or œdema of the scalp. She had not had any pain in the head or ears; the only discomfort had been some irritation of the conjunctiva of the left eye, which was probably due to facial palsy.

Results. When the patient was last heard from two years after the operation, the relief from headache and neuralgia continued, although, as previously stated, the pain for some time before the operation was of the most intense character. In other respects her general condition has been good. She still continues to be partially paralyzed, with astereognosis and sensory changes on the right side and still has paralysis in the distribution of the left seventh nerve. It is altogether probable that a lesion of some sort is still present; not improbably she has more than one lesion, as the symptoms point both to the pons and to the left parietal lobe. The case is especially interesting as showing how the cerebellopontile angle can be exposed and how pain and other symptoms of brain tumor are sometimes relieved even when full success is not obtained.

Case 4.—Tumor of the cerebellum or cerebellopontile angle. The tumor was not found, but great benefit was obtained by a craniotomy associated with excision of a portion of one lateral lobe.

History. C. E., aged twenty-three years, was admitted to the University Hospital, December 7, 1903, and was placed under the care of Dr. Charles K. Mills, who reported that his illness began several months prior to his admission to the hospital, the principal symptoms being dizziness and intense headache, starting in the occipital region and radiating toward the forehead. Five weeks later his sight began to fail and a week later some weakness of the left arm and leg were noticed.

At the time of his admission to the hospital he was suffering from the most distressing headache, which was almost constant, and from vertigo and ataxia to such an extent that it was almost impossible for him to walk alone or without support. Nor could he even stand alone unless leaning against some stationary object. His vision was so affected that he could not see gross objects and the examination of his eye-grounds revealed the presence of very marked choked disks and optic neuritis.[1]

Operation. The patient suffered so that it did not seem justifiable to postpone operation for further and more elaborate study of the case. Accordingly within a week of his admission to the hospital a left cerebellar craniectomy was performed by Dr. Frazier. The dura was noted to be unusually tense, and on reflecting the dural flap a considerable portion of the cerebellar hemisphere protruded through the opening. It was found to be almost impossible on account of the protrusion of cerebellar tissue to make further exploration, so the operator proceeded to remove from one-third to one-half of the lateral lobe. After this was accomplished, exploration was continued, but to no avail. No tumor could be seen or felt in any portion of the left cerebellar fossa.

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[1] This is Case 4 in Dr. Mills' paper, "The Diagnosis of Tumors of the Cerebellum and Cerebellopontile Angle, with Special Reference to Their Surgical Removal," New York and Phila. Med. Jour., Feb. 11 and 18, 1905.

Subsequent History. The postoperative record of this patient is one of unusual interest because of the remarkably rapid improvement which followed. Within one week of the operation the patient's headache had entirely disappeared; his vision was restored so that he could see minute objects on the ceiling as he lay in bed and his vertigo and ataxia had almost entirely disappeared. At no time during the convalescing period was the patient's condition such as to give any concern. He was kept under observation in the hospital for a period of two months, and inasmuch as there was not the slightest return of symptoms he was allowed to go home, with the understanding that he would return to the hospital if any of his cerebellar symptoms recurred.

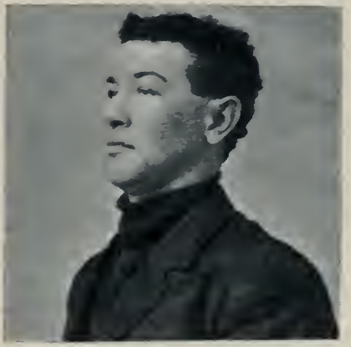

Fig. 3—Showing the general robust appearance of patient one and one-half years after decompressive operation for tumor of the cerebellum.

Present Condition. He returned to the hospital for an examination March 5, 1906. About two years and four months had elapsed since the operation and his headache, nausea, and vomiting have never recurred. His vision is practically normal and he is now engaged as a laborer in the Pennsylvania Railroad shops at Altoona. It should be said, however, that he occasionally has some attacks of vertigo His weight has increased to 184 pounds and he is in all other respects apparently perfectly well (Fig. 3).

Case 5.—Tumor of the cerebrum; decompression operation affording relief from headache for a period of three years.

History. W. D., aged twenty-two years, was admitted to the University Hospital, Nov. 5, 1903, having previously been under the care of Dr. W. O. Hermance. He was admitted to the service of Dr. William G. Spiller, by whom the following history was elicited: He has been suffering from headaches since 1897 and for the past nine months they have been especially severe. He has furthermore often complained of nausea, vomiting, and dizziness and his sight is completely lost.

Examination. On examination there was found to be no involvement of either the fifth or seventh nerves. The movements of the tongue and of all the limbs were unimpaired. The biceps tendon and the triceps tendon reflexes were not specially active. The grasp of each hand was normal, as was the resistance to passive movements in all the extremities. There was no involvement of the bladder or rectum and no ataxia of upper or lower limbs. There was no deviation from normal as to stereognostic conception in either hand, the gait was normal, there was no tendency to sway or fall, there was no Babinski reflex on either side and the patellar reflexes were exaggerated. There was persistent ankle clonus on the right but none on the left side. There was no word deafness and no aphasia. His sister reported that about three months previously he was unable to speak at all for at least an hour, but that this was the only time he ever entirely lost the power of speech. He was at times delusional, and imagined he was being put into a hole. He had what he calls "sinking spells," in which he felt weak and his heart stopped beating.

About six months prior to his admission to the hospital he complained of stiffness (?), probably weakness, in the back, left side of the neck, in the left arm and left leg. He had never had any convulsions, but from April to August of the present year, his whole body would get stiff for about half an hour. His memory is better than it used to be. He listens attentively to what is read to him and remembers it better than the persons who read it. The headache had been frontal and bilateral until February of this year and it seemed to have become more localized to the left temporal region.

Dr. Thorington, who had examined his eyes, reported that there were no ocular palsies, but choked disk in both eyes, slightly more advanced in the left. There had been numerous hemorrhages, large and small, old and recent. Examination of the urine on one occasion prior to his admission to the hospital revealed a few hyalin casts and a small quantity of albumin. Since the examinations at the hospital, after repeated examinations, only on one occasion was a suspicion of hyaline casts, at no time was there albuminuria.

November 5, 1903, Dr. Shumway made the following ophthalmic report: No light perception. Right eye, atrophy following choked disk. Nerve head much swollen, veins tortuous, no hemorrhages. Left eye, nerve head still more swollen, some atrophy, hemorrhage on the nasal side of the nerve, numerous white points of exudation.

6th. It was noted on this date by Dr. Spiller that pupils were dilated, there was no reaction to light and no ocular palsies. The ophthalmic reflex was present on each side. Patient closed the eyelids firmly and wrinkled the forehead well on both sides, the tongue was protruded straight, not wasted and there were no fibrillary tremors. His hearing was apparently not affected. He swallowed without difficulty. There was some tenderness to percussion over the left temporal region. There was no word deafness nor motor aphasia.

Upper Limbs. Movements of the upper limbs were free at all points. The biceps and triceps reflexes hardly obtainable on either side. Grasp of each hand and resistance to passive movements were normal. Sensation for touch and pain normal in all parts of the body, and the sense of position and stereognostic conception were unimpaired. There was no wasting.

Lower Limbs. Voluntary power normal, no hemiasynergy on either side and no wasting of the lower limbs. The sense of position was normal on both feet. The patellar reflexes were distinctly exaggerated on either side and more prompt on the left side. There was a slight indication of ankle clonus on the right side, although clonus was not as persistent as at the last examination. There was no clonus on the left side. The Achilles tendon reflex was exaggerated on each side, but more so on the left. The patient stood well with his feet together and the eyes open or shut. His gait was normal, although it was uncertain on account of his blindness. There was no Babinski on either side.

Further examination of the other organs including those of the thorax and abdomen proved negative.

Operation. Craniectomy was performed by Dr. Frazier under ether anæsthesia and an osteoplastic flap was reflected in the left frontal region. It was noted at the time that the skull and the dura were abnormally thick. There were no adhesions between the dura and the skull. Pulsation was visible and the brain did not bulge when the dura was reflected. Especially at the superior and anterior aspect of the opening the consistency of the brain seemed abnormally soft and an exploratory needle was introduced in various directions, but with negative results. The dural flap was replaced with sutures and the bony portion of the osteoplastic flap was removed for the purpose of relieving intracranial pressure.

Subsequent History. The patient remained under observation in the hospital until January 27, 1904, at which time it was noted that he had had no headaches since Dec. 28, 1903. His general condition had improved and he had gained in weight and strength. The last ophthalmic examination prior to his discharge from the hospital made Jan. 1, 1904, notes that the nerve is becoming more and more atrophied.

April 15, 1904. The patient was admitted to the hospital again with a history of having had no recurrence of headache after he had been discharged; from this time on the headaches recurred generally on the right side of the head in the occipital region, accordingly was it deemed advisable to perform a second palliative operation and on April 16, 1904, Dr. Frazier performed an osteoplastic operation, reflecting a flap from the right temporoparietal region, removing a flap 10 cm. broad and 7 cm. wide. It was noted at the time that there was no visible pulsation and that the brain appeared to be under considerable tension. The bone at the margin of the upper aspect of the opening appeared abnormally dark in color and unusually porous. An exploratory needle was introduced into the pons at about the middle of the opening but no fluid was detected.

Following the operation the patient complained for a few days of a severe supraorbital neuralgia which was attributed to the pressure which had been effected by the use of a pneumatic tube applied for the purpose of controlling hemorrhage. The patient was discharged from the hospital April 28th, about two weeks after the operation. His headaches had not entirely disappeared, although they were comparatively mild in character and the patient no longer required hypodermic administration of morphia to which he had been accustomed heretofore.

Present Condition, May 1906. The last observation was made two years and six months after the first operation and two years and one month after the second operation, and at this time it was reported that the patient had enjoyed almost absolute relief from his headaches. He is able to perform the duties connected with the management of a cigar store and with the exception of his loss of eyesight he appears to be entirely well. The optic neuritis was so far advanced when the patient was first seen that the possibility of saving his sight was out of the question.

Case 6.—Tumor of the cerebrum.

Patient. M. B., aged seventeen years was referred to the University Hospital, by Dr. R. Ross Jordan, Tyler, Clearfield County, Pa., and transferred from the service of Dr. Charles K. Mills, October 9, 1904.

History. He was struck on the back of the head nine months ago. He was not unconscious at the time he fell. About an hour later he became dazed, walking around aimlessly, remaining in this state for about half an hour. On Jan. 28, 1904, he was seized with shortness of breath, succeeded by tingling in the ears, and fell to the ground. It was said that during the convulsion his eyes were turned from side to side and that the whole body with the exception of the hands and wrists, fell into a state of spasm, lasting from five to seven minutes, with frothing from the mouth. Since that time he has had five attacks of this character, the later attacks having been preceded by violent occipital headache and vomiting.

Examination. He has marked optic nueritis, complains of intense headache and occasionally vomits. There is no hemiasynergia on either side. No paralysis of either facial nerve, the masseter, and temporal muscles contracting well on either side. Sensation for pain and touch is equal on both sides, so that there is no involvement of either the sensory or motor root of the fifth nerve. The extraocular movements are normal, but there is a lateral nystagmus when looking from either the right or left. The tongue is protruded straight, moves freely and there are no tremors. The head is not tender to percussion.

Upper Extremities. Grasp of each hand is equal and strong; sensation for pain and touch normal in each upper limb and it is the same on both sides. Stereognostic perception is normal in both hands. Biceps and triceps tendon jerk are not distinct in either side. There is no muscular atrophy. Resistance to passive movements is normal in both upper extremities. In the lower limbs no hemiasynergia. Resistance to passive movements normal, patellar reflex normal on each side, and there is no Babinski reflex. Sensation for touch and pain are the same on both sides, gait and station are normal with the eyes open or shut. There is not the slightest trace of hemiparesis or disturbance of sensation on the left side.

Ophthalmologic Report. From the service of Dr. de Schweinitz. Ophthalmic diagnosis hemianopsia. Pupils react normally to light and accommodation, the muscle balance normal; eye-grounds, media clear; O. D. marked optic neuritis, 5 D. no change in macula; O. S. swelling of disk 5 D; a few small fresh hemorrhages in the fundi. No change in the macula.

Operation. Dr. Frazier, Oct. 10, 1904. Inasmuch as the symptoms pointed entirely to a lesion in the occipital lobe, an osteoplastic flap was reflected exposing this region. The dura exposed in the opening presented a mottled appearance as though the seat of an organized traumatic or inflammatory exudate. There was slight visible pulsation and there seemed to be considerable tension. When the dural flap was reflected the brain immediately bulged through the opening even beyond the level of the scalp. From the opening a jet of yellowish-red fluid spurted three or four inches. About 45 c.c. of fluid were evacuated in this way. In order to close the defect it was necessary to reflect a flap from the pericranium inasmuch as the brain still bulged considerably through the opening. This was taken from the section of bone contained in the flap and the bone itself was removed. The wound was closed with interrupted silkworm-gut sutures and drain introduced through counter openings.

Subsequent History. October 13*th.* Since the operation the headache had entirely disappeared and the patient is now able to see objects which he could not before the operation.

24*th.* On the date of his discharge from the hospital it was reported that the patient felt better than he had for some months and that the vision of both right and left eyes had improved.

R. E. V. before the operation 6/30, at the present time 6/22.

L. E. V. before the operation 6/30, at the present time 6/15.

There is still a left hemianopsia and the swelling of the disks has decreased from 5 to 3 D.

Dec. 21, 1904. The patient was readmitted to the hospital. He returned to the hospital chiefly owing to the fact that the hernia cerebri had increased and especially for the past two weeks his vision had been failing. During the past two months he had gained forty pounds Examination of the eye-ground showed that the choked disk had entirely subsided, but that there was marked optic atrophy. There was left lateral hemianopsia for light only.

23*d*. The original field of operation was exposed and the dura was found intact. On reflecting the latter a canula was introduced and immediately a large quantity of blood-stained fluid escaped. Immediately after the evacuation of the fluid the hernial protrusion collapsed. The question arose at the time of the operation as to whether we were dealing with a cyst of the occipital lobe or the dilated horn of the lateral ventricle. No evidence could be detected of a neoplasm in the neighborhood of the operative field. The wound was closed in the usual way.

29*th*. Patient has made a satisfactory convalescence from the operation. There has been a free discharge of cerebrospinal fluid from the wound, requiring reinforcement of the dressing from time to time.

January 27, 1905. The patient was discharged from the hospital today, the wounds having healed *per primam* throughout. The hernia was not so prominent as it was before the operation. The patient's condition, with the exception of his impairment of vision, is entirely satisfactory. The optic atrophy which was discovered after the choked disk had subsided of course precludes the possibility of his recovering his vision. In a letter received from him, February, 1906, the patient reports that he is feeling very well, has had no bad spells, and is now in attendance at a school in Pittsburg, Pa.

Case 7.—Tumor of the left lateral lobe of the cerebellum. Decompressive operation, followed by striking improvement.

History. Inasmuch as this case was reported in full,[1] it will be necessary to refer here only to the salient points.

Patient. F. M., aged ten years, was referred to Dr. Frazier's service at the University Hospital by Dr. D. J. McCarthy, March 7, 1904. The essential symptoms in the case were a gradual increase in ataxia and pronounced headache, vertigo, and vomiting. There was a marked degree of choked disk on both sides.

Operation. *March* 13, 1904. Dr. Frazier removed the bone overlying the left lobe of the cerebellum and proceeded to explore the hemisphere for a tumor. Exploration failed to reveal the presence of a tumor and before closing the dura, one-third of the cerebellar hemisphere was removed. The effects of the operation were most gratifying. At his discharge from the hospital on March 27th, his condition was noted as greatly improved. His whole disposition had changed; he was bright and cheerful and complained neither of headache nor vomiting.

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[1] See Dr. Mills' paper "The Diagnosis of Tumors of the Cerebellum and Cerebellopontile Angle, especially with Reference to Their Surgical Removal," New York and Phila. Med. Jour., February 11 and 18, 1905, pp. 91–96.

Second Operation. November, 1904, the patient returned to the hospital because of the recurrence of some of his symptoms. On the following day the cerebellar fossa on the affected side was explored and an infiltrating tumor about the size of a walnut was found and removed.

The value of the palliative operation in those cases in which the tumor cannot be localized is well illustrated by this case. Between the date of the palliative operation and the time at which the tumor was finally found and removed the patient had been comparatively free from any subjective disturbance.

Case 8.—Suspected tumor of the cerebellum. Decompressive operation including removal of a portion of one cerebellar hemisphere, followed by considerable relief as to the vision, headache, vomiting, and vertigo. Death of the patient seven months later. No autopsy.

Patient. P. K., aged fifteen years, entered the neurological wards of the University Hospital in the service of Dr. William G. Spiller, May 9, 1905.

History. Eight months ago the patient complained of headache all over the head associated with vomiting. The attacks of vomiting were irregular, sometimes twice a day and again not oftener than twice a week; in the interval, however, he was occasionally nauseated. He complained for some time also of dizziness and difficulty in locomotion. He had never had any convulsions.

Examination. Dr. Spiller, May 12, 1905. Conjunctival reflex is distinctly impaired on the left side; corneal reflex normal, both cornea and conjunctiva are normal on right side. Movements of the face are normal on both sides and there is no involvement of motor or sensory root of the fifth nerve. Sensation for touch and pain is not impaired. Movements of the eyeballs in all directions are somewhat impaired, and somewhat inco-ordinate. Tongue is protruded straight, there is no atrophy and there are no fibrillary tremors. Hearing for the voice appears to be normal in each ear, speech and intelligence normal. When sitting up the patient holds his head fairly erect, possibly with a little tendency to incline it to the left.

The grasp of each hand is normal, finger to-nose test normal on each side. Neither biceps tendon reflex, triceps tendon reflex, nor the wrist reflexes are distinct on either side. Sensation for both touch and pain normal in both upper extremities. There is no wasting in the upper extremities.

In the lower extremities the voluntary movements are normal, and there is good resistance to massive movements. The patellar reflex is very prompt, even exaggerated on the right side, but not quite so prompt on the left side. There is no ankle clonus. The Achilles jerk on the right side is prompt, but not so prompt on the left side. There is no Babinski reflex on either side, the smaller toes being drawn downward, the great toes remaining still. Sensation for touch and pain is normal in each lower extremity, the cremasteric reflex is exceedingly prompt on the right side and prompt, but not so markedly on the left. The musculature of the lower limbs is poorly developed, the patient has some difficulty in standing. On standing with his feet close together, even with his eyes open, he usually falls to the left and backward. His gait is very ataxic with a marked tendency to go backward, and to the left. Hemiasynergy is not present on either side.

Provisional diagnosis, cerebellar tumor, more likely on the left side because of the tendency to hold the head to the left, to fall to the left and backward and because of the impairment of the left conjunctival reflex, and of the less degree of exaggeration of tendon reflexes on the left side.

May 12th, 1905. Ophthalmic report by Dr. Shumway. Completely blind in the right eye, left vision about 1/100. Both pupils are widely dilated. The right pupil does not respond to light and the left responds sluggishly. There is some limitation of the movements of the eyes, particularly of inward movement, the left eye not approaching within 5 mm. of the inner angle; the movements are very jerky in character. There is a high-grade optic neuritis of the right eye which is going into secondary atrophy of the nerve. The veins are very tortuous and there is a small hemorrhage on the outer side of the nerve and an elevation of 6 D. In the left eye there is also an optic neuritis and of a milder degree with an elevation of about 4 D. The left nerve is also becoming atrophic. The patient was referred to the surgical wards.

Operation. May 17, 1905. Under ether anæsthesia, Dr. Frazier performed a decompressive operation. The exposure of the left cerebellar hemisphere was attended with unusually free hemorrhage particularly from those sinuses which have been described on previous occasions as existing in the occipital bone in the neighborhood of the occipital protuberance. When the dura was opened the cerebellar tissue bulged considerably but on the surface of the hemisphere and to a depth of about one inch, no pathologic condition could be demonstrated. About one-third of the hemisphere was removed and the cerebellopontile angle exposed but with negative results. A very good exposure of the seventh and eighth nerves was obtained, and they were demonstrated to the eight or nine physicians who were witnessing the operation. No further exploration was made and the wound was closed.

Subsequent History. The patient reacted from the effects of the operation. Dr. Shumway reports that the optic neuritis had subsided very much, the apex of the swelling in the right eye is now only 3

D. as compared with the 6 D. before the operation, and the left eye 2 D. as compared with 4 D. before the operation. The nerves are becoming somewhat atrophied. The patient's vision has improved particularly in the left eye, so that he can count figures at a distance of two feet.

July 19th. Reported to-day that the patient can walk around the ward without any assistance, although still somewhat unsteady, but, however, no tendency to fall in any particular direction. He has now no headache, and his vision has been so improved that he is able to distinguish faces and objects.

No opportunity was afforded to make any observations after the patient was discharged from the hospital, and in answer to a letter of inquiry written in January, 1906, we learned that the patient had died December 24, 1905.

Case 9.—Suspected tumor of the cerebellum in which a decompressive operation entirely relieved the subjective symptoms.

History. S. L., aged twenty years, patient of Dr. T. H. Weisenburg and Dr. M. Radcliffe, was admitted to the University Hospital, Oct. 25, 1905, with the following history:

Family History. Father died of pneumonia. Mother is living and well. Two sisters and six brothers all in good health. No history of nervous disorders, tuberculosis, renal disease, cardiac disease or neoplasm.

Social History. Patient was born in Austria and came to this country when three years of age. Engineer by occupation. Uses alcohol and tobacco in moderation. Patient has a small urethral discharge at the present time of one month's duration. There is no syphilis.

Previous Medical History. Patient had measles during infancy; otherwise he has always been in excellent health. He was well until one year ago, when he began to experience nausea, followed by vomiting, with pain of a severe character, starting in the nape of the neck and shooting upward to the top of the head. At first these attacks were rather frequent, occurring every day or every other day, but they have greatly decreased in frequency. Two months ago the sight in the left eye began to fail and now he can distinguish only shadow and light. At about the same time he noticed that he was deaf in the left ear. On attempting to walk he feels giddy and staggers. This began about three or four months ago and has not increased much since then. Turning the head from side to side and backward causes some pain, but not severe. Lifting causes severe pain with blurring of vision. No motor paralysis.

Examination, by Dr. Weisenburg. Station with eyes open and shut good. Gait with eyes closed, good, though occasionally he has tendency to walk either to right or left.

Eyes. Pupils dilated with atropin. Convergence impossible with left eye, *i. e.*, on attempting to look at nose, the left internal rectus is weakened. Associated ocular movements to right and left are perhaps affected; *i. e.*, he can look either to right or left to the normal extent, but in a short time the eyeballs have a tendency to jerk back toward the median line. Upward and downward associated movements are normal. No diplopia. Ophthalmoscopic examination shows marked choked disk on both sides, especially the left. Both optic nerves are much swollen and hemorrhages are profuse. The appearance is that of an albuminuric retinitis.

The fifth, the seventh, and the twelfth nerves are normal. Hearing seems to be diminished on the left side. There is no trouble in eating; no dysphagia; no dysphonia. Memory is good. Both upper and lower limbs are normal in form. Reflexes and sensation are normal. There is no ataxia on either side. No involvement of bladder or rectum. No hemiasynergy. Touching conjunctiva on either side does not elicit any movement of eyeball while corneal reflex is present. There is no Babinski. The results of the examination of the nose, throat, and larynx by Dr. Grayson and of the sense of hearing by Dr. Randall were entirely negative.

Operation. Oct. 26, 1905. A decompressive operation was performed by Dr. Frazier, which consisted in a suboccipital craniectomy, the bone overlying the left cerebellar hemisphere was removed. There appeared to be an increased amount of intracranial tension and the dura did not present its normal pale, bluish color, but in most places and particularly in the upper and inner angle, it was yellowish, opaque and dotted here and there with whitish-yellowish specks. The dura was not incised.

The operative recovery was entirely uncomplicated. The wound healed throughout *per primam.*

31*st.* Dr. de Schweinitz made an ophthalmic examination and reported a right choked disk, plus 10 to 11 D.; left choked disk, plus 9 D. and no fresh hemorrhages.

November 3*d.* Dr. de Schweinitz reports O. D. choked disk, plus 11 D., some fresh hemorrhages. Vision 6/22. O. S. choked disk, plus 9 to 10 D., but more contracted laterally; fresh hemorrhages.

December 22*d.* Dr. de Schweinitz reported as follows: O. D. 6/12 subsiding neuritis, plus 7 D. O. S., subsiding neuritis, plus 7 D. It will thus be seen that choked disks have subsided about 4 diopters since the operation.

December 23*d.* The patient was discharged from the hospital to-day. Since the operation his vision has improved considerably.

January 29, 1906. Patient reported at the hospital for examination and said that there had been no headache, nausea, or vomiting. O. D. the disk can be distinctly seen, slight swelling of the edges, only about plus 1 D. O. S. disk apparently normal. This is one of the most striking effects on choked disk after decompressive operations that we have observed and is particularly interesting in that the results were obtained without opening the dura.

May 29th. The patient now has some headache, but much less than he had before the operation. He is occasionally dizzy. The patellar reflexes are a little increased. Gait and station are good even with eyes closed. Dr. de Schweinitz reports: Subsiding optic neuritis. R. E., marked loss of capillarity, especially temporal half disk swollen, 5 D. (eyeground 3 D.), hence actual elevation 2 D.; vessels shrinking; some exudate in macula. L. E., well-marked subsiding neuritis and postpapillitic atrophy.

Case 10.—Tumor of motor region.

History. P. S., aged twenty-five years, was referred to Dr. Spiller by Dr. de Schweinitz.

Family History. Father and mother living and well. No history of neoplasm, tuberculosis, liver or kidney disease. Cousin became insane and committed suicide. Mother says the whole family is very nervous.

Social History. Went to school until eighteen years of age, then studied law for three years, but was forced to give it up on account of his nervous condition, then became a fireman on a locomotive. Smokes to excess; drinks alcohol in moderation.

Note from Dr. de Schweinitz: "Double optic neuritis (choked disk), visual fields normal. No palsy of exterior muscles. Choked disk about 5 diopters."

Up to eight years of age had almost every infectious disease. When eleven years old he went to bed well one night and awoke blind and remained so for four months (treated at St. Agnes' Hospital). Sight gradually returned. According to his mother, "eyeballs were solid red, like clotted blood." Until seventeen years of age, he had fair sight and did not wear glasses. At seventeen, he began to be very nervous, and it was then that he began to take "weak spells." About once a month he would suddenly become weak, his knees would give way and he would fall to the ground, but would not lose consciousness. These fainting spells disappeared after a while and have not been present since he was eighteen years of age.

At twenty-one years of age he again wore glasses. His sight gradually diminished and he suffered severe pain in his eyes, especially behind the eyes. Temporary attacks of blindness, lasting a minute or a few seconds, occurred sometimes. He has always been a heavy smoker since he was seven or eight years old. He has averaged twenty to twenty-five cigarettes daily and also a pipe and a cigar a day. He had gonorrhea three years ago, but has not had a chancre or other venereal sore. He has been always subject to headaches, but within the past year, pain has been almost constant, worse at night, preventing sleep, and associated with attacks of vertigo, weakness, nausea, and vomiting. The headache is chiefly in the vertex, but sometimes occipital and above the eyes. He had a severe fall and struck on the back of his head when a child. Weakness disappears at will; if he "were to give in to it he would go in a heap." He has never had convulsions, but says that several times he has had attacks in which the whole left side, arm and leg, especially the leg, became numb, "as though dead;" the arm was only a little affected. He has no paralysis or paresis anywhere. He was a fireman on a locomotive three years and was so nervous that he gave up the work. Every little noise would make him jump, and he was always expecting something to happen, "lost his nerve," as he expressed it. Masturbated freely before sixteen years of age.

Examination. Sensation seems normal everywhere and to every form of stimulation, and there is no astereognosis and no loss of muscle sense. Ocular movements are good. Slight nystagmoid movement is seen on extreme lateral excursion, especially to the left. Reflexes are normal. There is no Babinski and no ankle clonus.

Cardiac condition: Irregular heart action without marked accentuation of either aortic or pulmonic sounds.

Patient states that the attacks of stiffness of left lower limb previously mentioned are usually preceded by much greater headache, a "sensation of something inside expanding and bursting out." They frequently occur at night and in them the left leg becomes very rigid and is drawn across the right, but it can be voluntarily moved. The left arm is to a less extent involved and still less the left face. During the attack, consciousness persists, but the whole left side feels as though "dead." He has almost constantly a blowing tinnitus aurium; only on the right side. He quit railroading in November, 1905, and since then has had only one or two attacks of spasm; previously had one or two weekly.

March 20, 1906. Yesterday morning, in getting out of bed he had headache. Between 10 and 12 o'clock he became weak in his knees, but did not fall. In the afternoon he vomited and complained of intense headache and became unconscious. The left arm was flexed on the forearm and was stiff. The left lower limb was stiff and probably the left side of the face was affected. He was unconscious all night and very noisy and hard to restrain. This morning he is perfectly conscious, but in an agony of headache, crying out at times and tossing about in bed, clutching at any one near him and begging for relief. There is no motor weakness in any of the limbs, but he has some slight disturbance of sensation in the right hand. Stereognostic perception, the sense of position and tactile sensation are normal in each hand. The spasms which he has had have been in the left side and have always been tonic (never clonic). The patellar reflex is exaggerated on the left side and possibly slightly so on the right. Achilles tendon reflex is not exaggerated and there is no Babinski reflex.

Operation by Dr. Frazier. March 20. 1906. Under ether anæsthesia the usual motor opening was made, the flap measuring 3½ x 4 inches in width and length. When the osteoplastic flap was reflected it was noticed that at a point about 2½ inches from the front of the posterior margin of the opening and just below the superior opening the dura had already been punctured by the neoplasm, and immediately overlying this point the bone had been almost entirely worn away by the tumor. There was no visible nor palpable pulsation. The exposed dura was of a greenish hue and gave the appearance as though there were beneath it numerous large vascular channels. There was a peculiar soft fluctuating sensation communicated to the examining hand. An exploratory incision was made in the dura, beginning at the point where it had ruptured spontaneously. A tumor was immediately exposed, apparently of the infiltrating character and filled with large vessels. Its removal was considered impracticable. The dural incision was closed and the lower half of the bone contained in the flap as well as a considerable portion of the temporal bone were removed. The wound was closed as usual with interrupted silkworm-gut sutures and the drainage introduced through two counter openings.

21*st*. The headache, of which he complained so bitterly before the operation, has entirely disappeared. Patient can move his left leg, but has no movement in his left arm, forearm, or fingers. Sensation is not impaired. There is some impairment of the left facial nerve.

22*d*. The movements of the left lower limb are still unimpaired. The left upper limb is completely paralyzed, as is also the lower part of the left side of the face, the muscles supplied by the superior branch of the facial nerve being weak. The tongue deviates a little to the left when protruded. The left biceps and triceps reflexes are not distinct; the left patellar reflex is exaggerated. There is no ankle clonus on the left side.

Subsequent History. March 23*d*. Drainage removed to-day and wound in excellent condition.

25*th*. Dr. de Schweinitz reports that in the right eye there is optic neuritis, a greenish-white swelling confined to the disk and covered with numerous particles, probably cholesterin. There are no hemorrhages. Swelling of the disk is plus 3 D. In the left eye the conditions are precisely similar; swelling of disk, plus 4 D. Vision O. D. 4/25, O. S. 4/12.

26*th*. Patient's general condition is excellent. The dressing has been removed and he is up and about the ward.

27*th*. For the first time the patient can lift the left arm and raise his hand over his head without any difficulty; he can flex his fingers.

30*th*. Patient still has some weakness in the lower side of the face, the left upper arm is still weak, although power is gradually returning. Tactile sensation, sense of position, stereognostic conception are normal in the left hand. He is able to walk unaided.

April 1*st*. Dr. de Schweinitz reports, under date of to-day, optic neuritis in right eye plus 3 D., in left eye, plus 4 D. O. D. V. equals 4/20 (?), O. S. equals 4/10 (?). Vision is very much blurred. Evidence of temporary blindness.

April 4th. The patient was discharged from the hospital to-day, having had absolutely no recurrence of headache.

Case 11.—Tumor of cerebellum.

History. E. F., aged sixteen years, was referred to Dr. Spiller, July 16, 1904, by Dr. J. S. Baer and Dr. R. G. Curtin. The father of the patient possibly had had syphilis. The menses ceased about two years ago; having begun when she was fourteen years of age, they had, therefore, been present only a short time. For about one year she had had a severe sensation of pressure at the back of the head, as though someone were pressing her brain out, but this sensation is not felt now. She has had headache, not localized to any one part of the head, and has had "neuralgia" in the left side of the forehead about a year. Before December, 1903, the headache was severe. About a year ago she began to have nausea and vomiting, at first about every six weeks, but these symptoms gradually became more frequent until they occurred every two or three days. The vomiting had no relation to eating. About a year ago she began to be dizzy, but has not been dizzy since February, 1904. Dr. Risley has recently discovered optic atrophy, secondary to choked disks. She has never been unconscious. She has had difficulty in lying on her right side, but this sign seems to have disappeared. Lying on the right side caused her head "to kind of draw." She has never had convulsions. Sight has been failing about two years. Difficulty in walking was noticed about November 1903; she staggered, and the straggering seemed to be more to the left.

Present Condition, July 16, 1904. There is no involvement of either seventh nerve; masseter contracts well on each side, tongue protrudes straight, not atrophied and shows no fibrillary tremors. Movements of eyeballs are free in all directions, but distinct nystagmus is observed in turning the eyes far to the left or right, and probably is more intense in turning the eyes to the right. The right pupil may be a trifle larger than the left. The reaction of the pupil to light is preserved; also in convergence. Accommodation cannot be tested because of poor sight. She can count fingers with either eye, but the sight is more impaired in the left eye. She hears a low-ticking watch one or two feet from either ear, and has not noticed any deafness. She has had buzzing in both ears, especially in the left, but Dr. Baer states that she has had accumulations of wax in the ears. The face is somewhat puffy and expressionless. The exit points of both fifth nerves are sensitive, but the left supraorbital foramen is more sensitive than the right. She now holds her head straight, but says she formerly inclined her head to the left. Sensations of touch and pain are normal in each side of the face.

Upper Limbs. All the movements are free; the grasp of each hand is normal. The biceps and triceps reflexes are not distinct on either side. Sensations of touch and pain are normal in each upper limb. The finger-to-nose test with eyes closed does not show distinct ataxia.

Lower Limbs. Voluntary power is normal. The patellar reflex may be a little prompter than normal on each side. She staggers a little when standing erect with eyes closed, and the gait is uncertain, with a tendency to stagger more to the left. The gait is cerebellar in type, but the staggering is not intense. The Achilles jerk is not distinct on either side, nor is the Babinski reflex. Sensations of touch and pain are normal in the lower limbs.

Dr. W. H. Powell first examined the eyes in November, 1903, Vision of the right eye, 20/50; of the left eye, 20/50. Pupils react to light and accommodation. Peripheral vision normal; media clear, but fundus suggests a low grade of neuritis. In July, 1904, vision of the right eye was 20/100, of left eye 4/200, and she had optic atrophy and markedly contracted fields.

Chiefly because of the history of probable syphilis in the father, Dr. Spiller employed mercury and iodide, and by Aug. 16, 1904, her gait had become less ataxic. By May 5, 1905, headache had disappeared, but about once a month she still had a drawing sensation, as though the brain were being pulled out. Ataxia was slight. The patellar reflexes were about normal.

February 22, 1906. Dr. de Schweinitz found typical postpapillitic atrophy.

Operation. March 9, 1906. Suboccipital craniectomy was performed by Dr. Frazier under ether anæsthesia. The operation consisted in removing that portion of the occipital bone overlying the left cerebellar hemisphere.

Subsequent History. March 28, 1906. Since the operation was performed the condition of the patient has improved, but only to a moderate degree. The pain referred to the arms has been very much less severe and at times she has had none at all. Her headaches also have been less severe and less frequent. Although confined to bed for the past year, she is now able to get up in a wheel chair. Although the dura was neither incised nor removed at the operation, there is quite marked bulging over the left cerebellar hemisphere, showing that the dura has yielded to the pressure from within and that it is possible for a hernia to develop without opening the dura.

Second Operation. April 4, 1906. After consultation with Dr. Spiller it was decided that, owing to the fact that the headache and pain in the arms had not been entirely relieved, it would be advisable and afford greater relief to the increased tension to split the dura. Following an injection of $\frac{1}{6}$ gr. of morphine, and under ether anæsthesia, the left cerebellar hemisphere was exposed by a muscle-splitting operation. A vertical incision was made through the middle of the original flap, the edges of the wound retracted and a crucial incision made in the dura about one and one-half inches in either direction. The cerebellar tissue bulged considerably through the opening, but the exposed tissue was normal in appearance. The musculo-aponeurotic layers of the wound were closed with catgut sutures and the scalp with interrupted silkworm-gut suture.

April 16th. The following notes were made by Dr. Spiller: The paralysis in the lower distribution of the right facial nerve supply is distinct, although the upper distribution of the nerve does not seem to be affected. She has slight weakness of the right upper and lower limbs. The patellar reflex is prompt on each side, but prompter on the right, and there is slight tendency to ankle clonus on the right side. The Babinski sign is present on the right side, but uncertain on the left side. There is no disturbance of sensation for touch or pain in any part of the body.

May 23d. For the first few weeks after the second operation the patient had occasional headaches and pain in the upper extremities. For the past two weeks, however, she has been almost entirely free from both these symptoms. She has gained in strength and is able to be up a considerable portion of the day. A hernia cerebelli has increased somewhat since the second operation (Fig. 4). She has recovered sufficiently from the effects of the operation and is sufficiently relieved to warrant her being discharged from the hospital. Her gait is still ataxic. She menstruated in May, 1906, the first time within a period of four years.

Case 12.—Cerebellopontile tumor. Decompressive operation, followed by disappearance of headache, vomiting, and by subsidence of choked disk.

History. E. D., aged twenty-four years, was referred to Dr. Frazier by Dr. Sherman Voorhees, of Elmira, N. Y. She was seen in consultation also by Dr. Spiller. About two years ago the patient noticed that her limbs were weak, that she would stagger in walking, although she did not feel dizzy. About one year ago she began to notice that her vision was failing. At this time she would have attacks, when for a few minutes everything before her would seem black; following the attacks she could see perfectly well again. At the same time she began to have attacks of nausea and vomiting, and noticed that she was becoming slightly deaf in her right ear. For the past three months she has had considerable headache, sometimes dull, at others, sharp and shooting in character. At the time of her admission, Feb. 16, 1905, she is practically blind and unable to walk.

Examination, February 17th. It was noticed that in showing her teeth, the right corner of the mouth is not drawn up nearly so well as the left, and in drawing up each corner separately, the movements on the right side are distinctly less than the left. On closing her eyelids forcibly the resistance to passive movements is less on the left side than on the right side; the masseter muscle seems to contract equally well on one side as on the other. The ocular movements are normal and there is no distinct nystagmus. There is no disturbance of pain and tactile sensation in the face, although she complains of a "queer" feeling on the right side of the face and right side of the mouth and of the face feeling numb, as though it were asleep. Further examination gives the impression that both the left temporal and masseter muscles contract a little more than the right, although it was subsequently found that on both sides the muscles respond equally well to Faradic current. The tongue is protruded straight and there are no fibrillary tremors. The right eyelid seems to be slightly more prominent than the left.

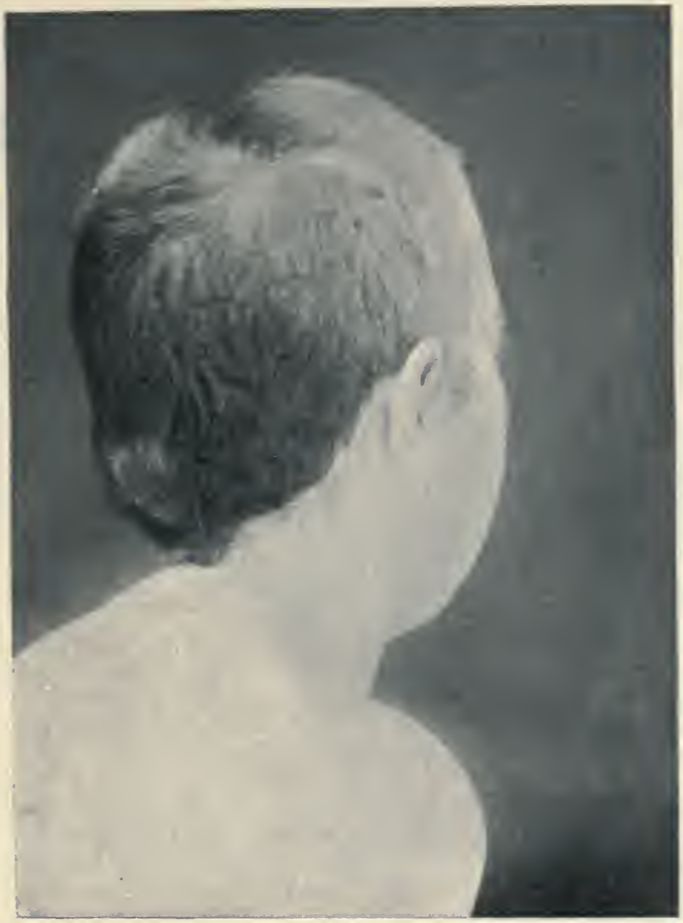

FIG. 4.—Showing a hernia cerebelli after a unilateral suboccipital craniectomy. The patient prior to the operation had been bedridden for over a year, is now able to be up and about and has been relieved almost entirely of her pressure symptoms.

In the upper extremities the grasp of each hand is normal. Biceps reflex is normal on each side; the triceps reflex and wrist reflexes are not distinct on either side; sensations for touch and pain are normal on both sides. There is slight ataxia on each side, a little more on the right.

In the lower extremities the patellar reflex is a little prompter than normal on both sides There is no ankle clonus. The Babinski sign is uncertain on each side, as she resents any attempt to irritate the sole of the foot. The Achilles jerk is absent on both sides; there is no hemiasynergy and no weakness of any of the limbs; gait is slightly ataxic, specially noticeable when she turns; in walking the patient staggers more to the left than to the right. She staggers when her feet are together almost as much when her eyes are open as when they are closed.

Examination of Ear. Dr. Randall finds little hearing in the right ear by air or bone, except for the Galton whistle. Tests are slightly contradictory, but indicate middle ear trouble on the right side, complicating semi-complete right acoustic paresis. Hearing on the left side is not impaired.

Ophthalmic Examination by Dr. Shumway. Pupils are dilated; right pupil responds when light is thrown in from the temporal side, in which position it is recognized. The left pupil does not respond to light and the eye is blind. There is choked disk on both sides; elevation on the right side is about 6 D. and on the left 7 D. The nerves are beginning to atrophy. The eye movements are carried out in a jerky manner, the muscles acting feebly, and the eyeballs return to primary position immediately. There is no paralysis of the ocular muscles.

Operation. February 23*d.* Under ether anæsthesia Dr. Frazier performed a suboccipital craniectomy, removing the bone overlying the right cerebellar hemisphere. Owing to the fact that bleeding was so profuse, it was decided to discontinue the operation at this stage until the patient had fully recovered from its effects.

A radical operation was performed two months later and was followed by death within five days (Figs. 5 and 6). After the decompressive operation the choked disk subsided from 7 D. to 3 D., although unfortunately, the optic atrophy was so advanced that it precluded the possibility of restoring the vision. The hearing improved somewhat after the operation and the nausea and vomiting ceased. Headaches disappeared almost entirely and the patient was able to get up and walk about the ward with but little assistance.

Case 13.—Inoperable tumor of brain. Decompressive operation, followed by little or no amelioration of the symptoms, although prolonging the patient's life.

Patient. R. C., aged thirty years, was referred to Dr. Charles H. Frazier by Drs. Thomas O. Nock and Wharton Sinkler, March 15, 1906, the latter having seen the case in consultation.

History. There was a history of the patient having had an attack of vertigo while walking about three months ago, and had she not been caught by her husband, she would have fallen to the ground. At the same time she had some numbness in the left upper and lower limbs. Since that time she has had two attacks of numbness of the limbs, but no vertigo. She has had more or less persistent headache for the past year, occurring about three or four times a week, usually in the mornings. There have been no convulsions. Three weeks ago the symptoms became much more severe, headache more intense and numbness in the left extremities more marked. At this time it was noticed that the muscles of the left side of the face were gradually becoming weak, the muscles becoming paralyzed within a period of from four to five days. Up to this time there was no weakness in the extremities, but about a week after the involvement of the facial muscles was noticed the grasp of the left hand became weak, and about the same time weakness was noticed in the left lower limb. The upper extremity sometimes is flaccid, at other times spastic, but the lower extremity has never been spastic. The pulse has varied between 44 and 60. The respirations have been alternately shallow and moderately full, the respiratory rate ranging from 16 to 30 a minute. For the past four or five days her temperature has been subnormal. About eight days ago, for the first time, the patient became stuporous for variable periods of time. These periods of stupor have gradually increased, and during the past two days the patient has been almost comatose. This condition of mental hebetude was first noticed about two weeks ago.

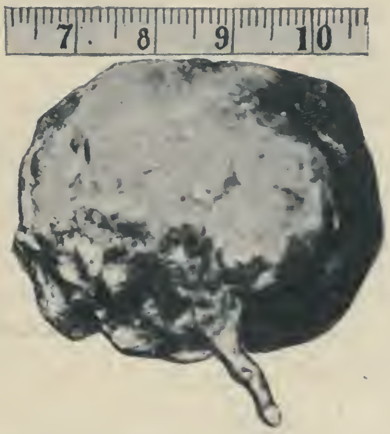

Fig. 5.—Photograph of fibroma removed at operation from the cerebellopontile angle.

Examination, March 15, 1906, *by Drs. Nock, Sinkler, Frazier and Spiller.* Stupor is so great that the patient can be roused but slightly. She lies in bed with her head turned to the right, but there is no deviation of the eyeballs. There is no tenderness on percussion of the scalp. She is able to move the eyeballs well to the right, although there may be slight impairment of the left eyeball to the right, but probably not of the right eyeball to the left. The right pupil is larger than the left. Neither pupil reacts to light. It is impossible to tell whether there is any deviation of the tongue or to test the muscles of mastication.

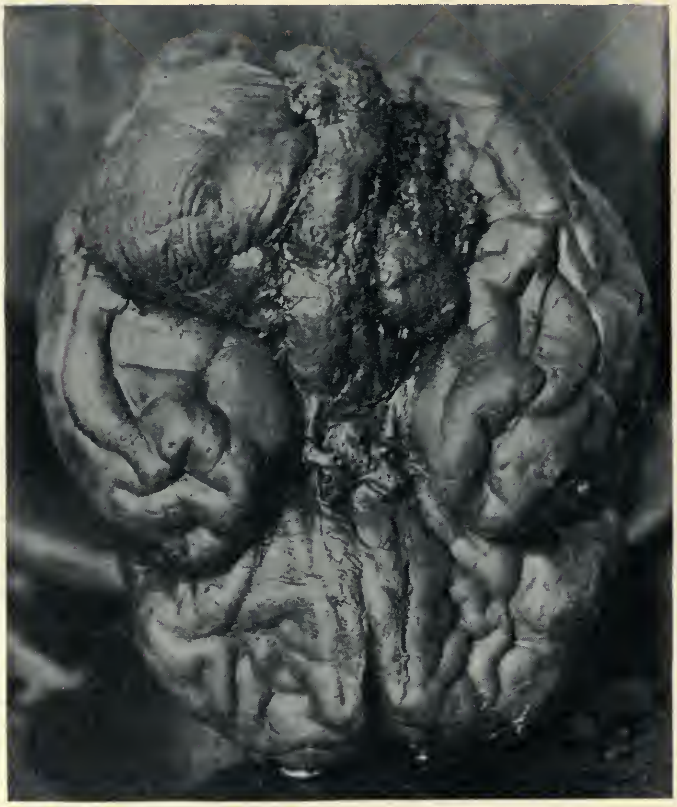

Fig. 6.—Photograph of brain showing portion of tumor remaining in the cerebellopontile angle after operation.

Left Upper Extremity. The left extremity is very weak, although she has a little voluntary power in it and can flex the limb feebly at the elbow. She cannot make a fist with the left hand. The biceps tendon and triceps tendon reflexes are present, but weak. Sensation is not disturbed.

Left Lower Extremity. The lower extremity is very weak, although she can move it slightly. The patellar reflex is present on the left side, but not very prompt. The Achilles tendon reflex is present on each side and about normal. There is no ankle clonus and the Babinski sign is very typical on the left side, as it is on the right, although not so pronounced. Pin-prick is perceived in the left lower limb.

Examination of the Blood. Systolic pressure, 165; hæmoglobin 65 per cent.; white blood corpuscles, 21,240.

Urinalysis. There is slight albuminuria, no sugar and a few granular casts.

Ophthalmic Record by Dr. de Schweinitz. This examination was made a week or more before the patient came to the hospital and disclosed a moderate optic neuritis of each eye, most marked on the right side. There are a few hemorrhages in the neighborhood of the optic disk. The neuritis appears to be in an early stage of its development; on the left side it is just beginning. The pupil reacts normally. There appears to be a divergent strabismus, usually more marked on the right side and probably dependent on paresis of the right internal rectus. Some examinations appear to indicate that there was failure in the movement of the eyeball upward, therefore some involvement of the right superior rectus. This was not certainly determined, as there did not appear to be much difference between the two sides; both lids droop to about an equal extent, but the droop is one probably due to weakness and not to paresis. The visual field, roughly taken by the hand, appeared to be undivided; at least there was no hemianopsia

Operation. March 15, 1906. The date the patient was admitted to the hospital an osteoplastic operation was performed, in which the motor region was exposed. It was noticed when the flap was reflected that there was some bulging of the dura at the lower and posterior angle of the opening. An exploratory incision about one and one-half inches long was made at this point, at which the dura seemed very much thinner, as though undergoing atrophy from pressure. The brain contents bulged through the opening thus made, and an exploratory cannula was introduced for about one and one-half inches, as there was some reason for suspecting the presence of an abscess. It seemed as though the cannula entered a cavity; at first some brownish fluid flowed from the cannula and afterward pure blood. The exploratory incision was made about one-half inch behind the anterior margin of the opening and a small section of tissue removed for examination. Concluding the lesion was a subcortical one; we decided to discontinue any further exploration. A considerable portion of the bone in the flap was removed and the dural incisions and the incisions in the scalp closed.

From the date of the operation until the time the patient left the hospital, ten days later, the patient manifested but little, if any, improvement as a result of the operation. There were times when she seemed to be a good deal brighter than she had been in the immediate past, and also times when she had a little more movement in the left upper extremity. Mental condition, on the whole, was better, but apart from this there was very little, if any, change. It might be noted that the leukocytosis, which existed before the operation, continued afterward, and at one time the white blood corpuscles numbered 36,000. In a differential count that was made March 25, 1906, there were 94 per cent. of polymorphonuclear cells. The persistent leukocytosis in the absence of any wound infection was strongly suggestive of the presence of an abscess.

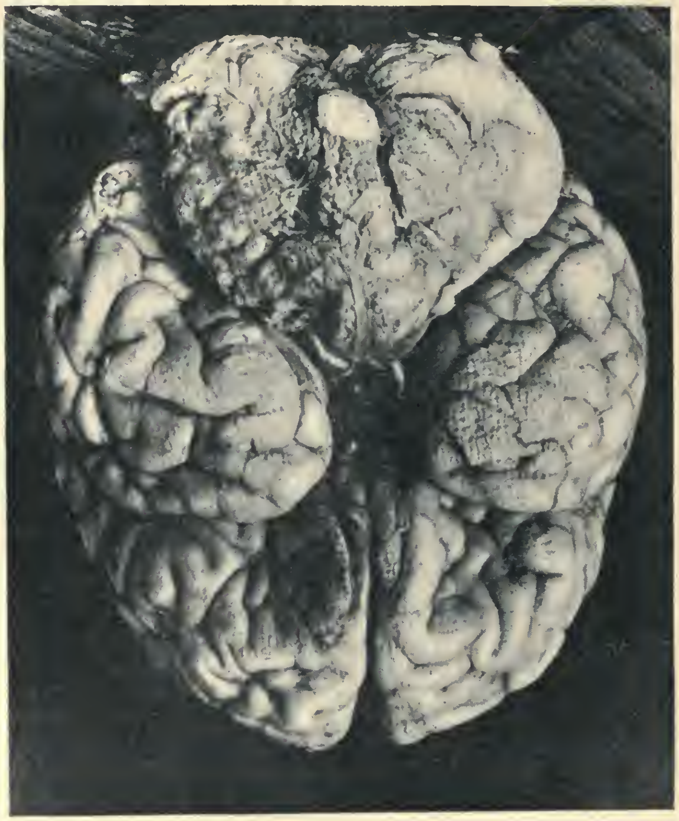

Fig. 7.—Tumor of the cerebellopontile angle.

Postoperative History. May 24, 1906. A letter recently received from the patient's physician, Dr. Nock, says that her present condition is about the same. There is complete paralysis in the left upper and lower extremities and now gradual weakening of the muscular power in the right arm and leg. When awake the patient talks intelligently and is interested in what is going on, although there is a tendency toward confusion of ideas at times. Her appetite is good and she takes enough food to maintain her nutrition. Her temperature varies from 99 to 99.3. Pulse is regular.

We include the following case, which was in the service of Dr. Mills and Dr. Martin and was studied also by us:

Case 14.—Stafford, male, aged forty-six years, was under the care of Dr. C. K. Mills in the University Hospital, in 1902. He was seen also by Dr. Potts and Dr. Spiller

The patellar reflexes and Achilles tendon reflexes were lost; the gait was ataxic. Headache, dizziness, optic neuritis, nystagmus and paræsthesia of the left side of the face were present among other symptoms. The diagnosis of cerebellar tumor was made. A palliative operation over the cerebellum was performed by Dr. Edward Martin in 1902. The patient's condition was much improved. He died in 1906 and a tumor was found in the left cerebellopontile angle (Fig. 7).

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PROGRESSIVE MUSCULAR ATROPHY OF CERVICOBULBAR TYPE OCCURRING WITH CERVICAL RIB.*

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Hugo Levi[1] refers to a case reported by Borchardt in 1901, in which in association with bilateral cervical rib, symptoms regarded by Oppenheim as those of syringomyelia were found. Borchardt concluded that cervical rib may occur with diseases of the nervous system, especially syringomyelia. Oppenheim mentions a second case of syringomyelia with cervical rib observed by him. Levi gives the history of a case in which the symptoms of multiple sclerosis were found associated with cervical rib. He refers to Helbing's statement that scoliosis is almost constant with cervical rib. In the case that we report lordosis was present.

Levi acknowledges that multiple sclerosis in his case may not have had a close connection with cervical rib, but as many regard each of these conditions as a congenital defect, some relation between them may have existed. Oppenheim has advanced a similar view in regard to the association of syringomyelia with cervical rib. I have recently had

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* Read before the Section on General Medicine of the College of Physicians, March, 1906.

[1] Hugo Levi, *Neurologisches Zentralblatt*, November 1, 1904, p. 988.

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the opportunity to study a case, which presented the symptoms of cervicobulbar palsy associated with cervical rib, and without sensory disturbances. The

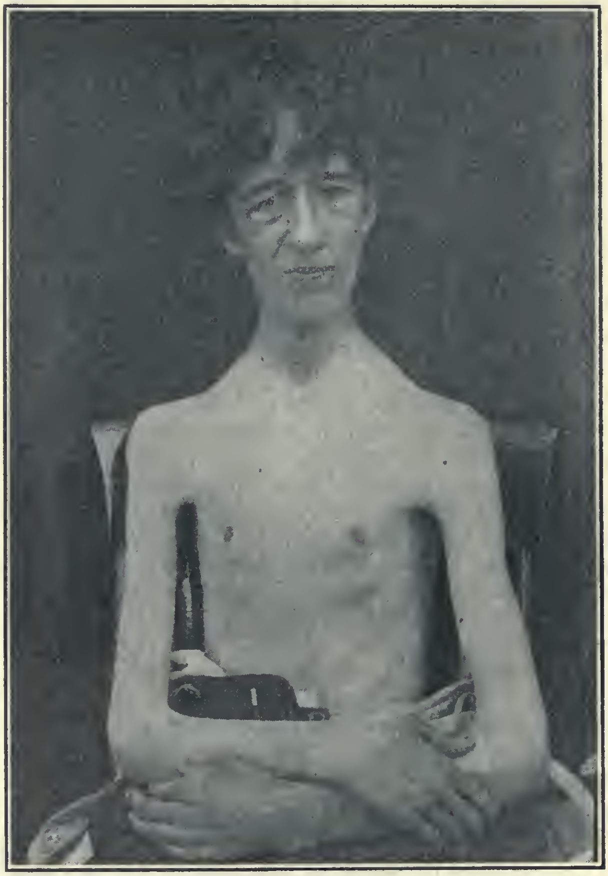

FIG. 1.—Showing great atrophy in the lower part of the face, the shoulders, and especially the upper part of the upper limbs.

occurrence of such a palsy in so young a person is extraordinary, and makes the case one of unusual interest. We may assume that some defect of the central motor system was present from the embryonic period in this case, and that the cervical rib was merely another congenital anomaly. It is well known that where one congenital malformation is present careful search may reveal others. Every effort was made to obtain a necropsy in this case, but without success. The patient died a few months after examination. The symptoms indicate a progressive atrophy of the cells of the anterior horns of the cervical region and of the motor bulbar nuclei with probably little implication of the pyramidal tracts.

Two cases of syringomyelia (Borchardt, Oppenheim), one of multiple sclerosis (Levi), and one of cervicobulbar palsy (Spiller and Gittings) associated with cervical rib have been observed. These are all diseases that are supposed to result from faulty development of the central nervous system, and such as might occur with anomalous developments elsewhere in the body.

The notes of our case made December 10, 1904, are as follows:

G., nineteen years old, is the younger of two children. He was healthy until about six months ago. He never was very stout. He has not had any pain except in the back of the neck, but this has not radiated into the shoulders, and was probably a result of the atrophy. When he was nine years old he had a severe fall on the pavement, was unconscious, and had convulsions, but has not had any convulsions since. He has not had headache. He has smoked much, and has been a great swimmer. He is very irritable and complains of much difficulty in swallowing; he has had many falls. Speech has been peculiar, he talks with great difficulty, and his speech is very indistinct and bulbar in type. He is not aphasic and can say what he wishes to. The tongue is protruded straight, is greatly atrophied, especially on the left side, and presents marked fibrillary tremors. The soft palate is almost immovable. The masseter muscle on each side contracts, but the contraction is not very strong. Saliva dribbles from the mouth. The jaw jerk is not very prompt. He cannot blow out a lighted match. He separates his lips with

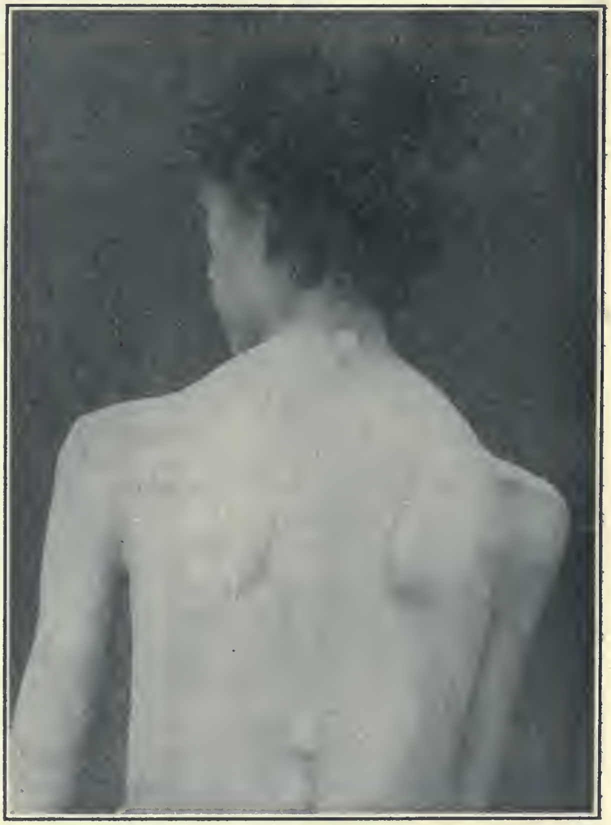

FIG. 2.—Showing the prominence of the scapulæ.

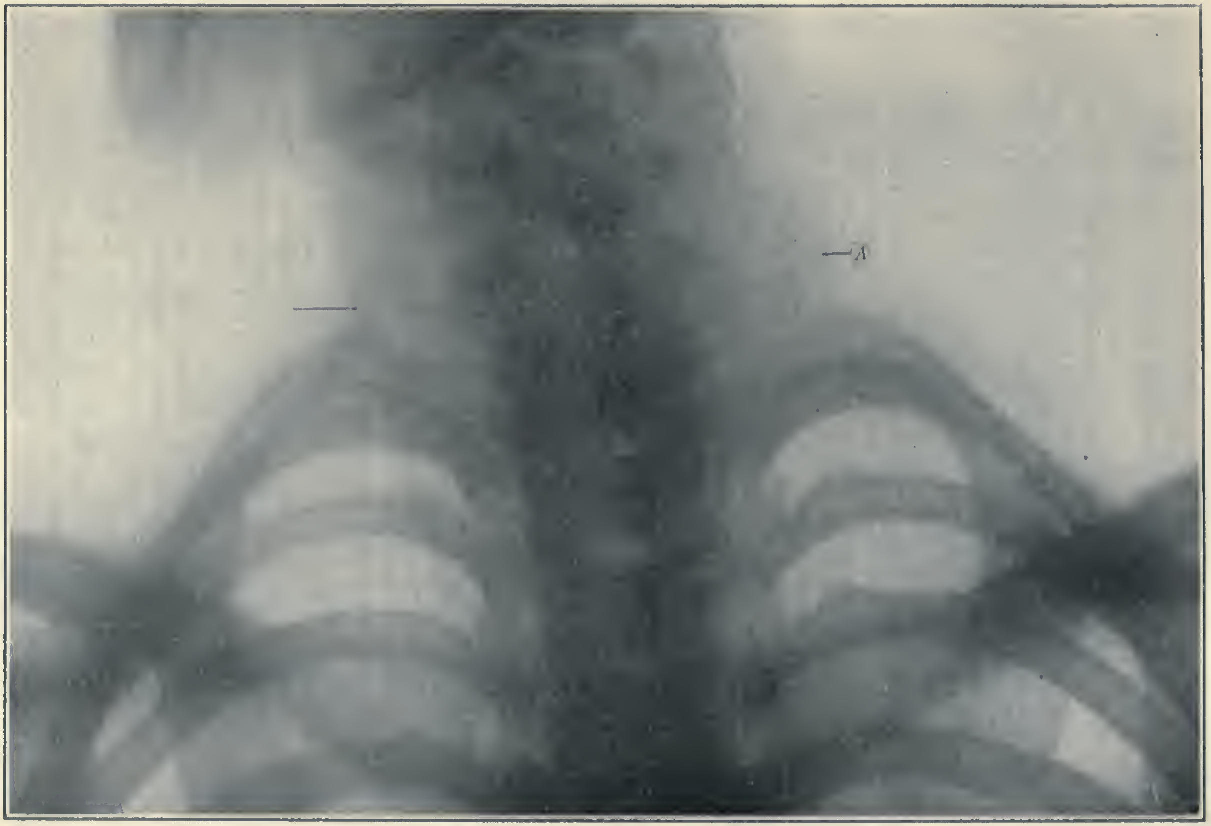

FIG. 3.—Bilateral cervical rib, more pronounced on one side. Not so distinct in the reproduction as in the photograph.

difficulty, when his teeth are placed together, and while he can draw up each corner of the mouth imperfectly, he does not draw up the left corner quite so well as the right. He closes the eyelids firmly, and wrinkles the forehead well on each side. The cheeks are extremely atrophied, and there is very little flesh on the lower part of the face. Sensations of touch and pain in the face are normal, as are also the extraocular muscles. The iritic reflex to light is prompt. When his head is moved backward he has pain directly over the upper part of the spinal column. He can bend his head backward and forward and to the left, but cannot turn it to the right. There is no tenderness to pressure over the upper part of the neck. The vertebræ are prominent, and the muscles of the neck are atrophied. Hearing as tested by a watch seems to be normal in each ear.

The upper limbs and trunk are greatly wasted. He cannot raise the right upper limb at the shoulder at all, and can raise the left only to a right angle with the trunk. The biceps jerk is very weak on each side. The right upper limb in the upper part is more wasted than the left. He has no triceps reflex on either side, no biceps reflex on the right side, and very little on the left. He cannot flex the right forearm on the arm, but can flex the left. The grasp of the right hand is feeble, and that of the left hand is good, though not normal. The scapulæ stand well away from the trunk, leaving a deep depression beneath each. The vertebral column is curbed backward in the thoracic and cervical portions. Sensations for touch and pain are normal in the trunk and upper limbs. Fibrillary tremors are not distinct (at a later examination, December 30, 1904, they were very distinct in both upper limbs). The lower limbs are much better developed than the upper, but are somewhat wasted. Voluntary power and resistance to passive movements in the lower limbs are good. The patellar reflex is prompter than normal on each side, the Achilles reflexes are about normal. Babinski's sign is not present. Sensations for touch and for cold are normal in the lower limbs. He walks up and down stairs, dances and seems to have little or no loss of power in the lower limbs.

Dr. Grayson made an examination of the throat on

December 30, 1904, and reported: "Pronounced paresis of palatal muscles on both sides, possibly a little more on the right side, also some atrophy of palatal muscles on the right side. Paresis of upper pharyngeal muscles. Complete paralysis of the epiglottis. Sensation for touch in the pharynx and soft palate is apparently normal."

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ADVANCES IN THE SURGERY OF THE NERVOUS SYSTEM CONSIDERED FROM THE STANDPOINT OF THE NEUROLOGIST.*

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(From the Department of Neurology, University of Pennsylvania.)

Some surgeons when writing upon this subject are wont to express their amazement at the truly marvelous advances which have been made in the surgery of the nervous system during the past one or two decades, and are given to expressions of admiration for the skill exhibited by members of their own craft, acknowledging, however, the debt they owe to their neurological brethren.

Neurologists, being no less generously inclined, while admitting the valuable services of the surgeon, are prone to subscribe to the views of the famous Roman physician Galen, who, in 200 A. D., expressed himself of the opinion that surgery is only a method of treatment and surgeons the instruments. The modern neurologist would go even a step farther and would carefully select the surgeon to do his operation. In these days of specialists, when surgeons limit themselves to operations upon certain portions of the body, it is a source of regret that the exclusive practice of surgery of the nervous system does not gain more advocates.

Most surgeons believe themselves capable of diagnosticating appendicitis, and of operating upon such

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a case. The pathology of the condition to them is, or should be, an open book. How many of the surgeons who operate upon the brain are capable of an accurate diagnosis, and how many know the pathology, to say nothing of the anatomy and physiology of the nervous system? And yet some surgeons never hesitate to act on their own initiative and without neurological assistance. It is this want of knowledge which has caused the pessimism of some surgeons and made possible the many atrocious operations of the past, and some which are practised even in the present day.

The surgeon should know at least enough of the anatomy, pathology, and physiology of the nervous system to give the patient falling into his hands the best chance possible for life and health; or he should seek the aid of the neurologist to this end. It must not be forgotten, however, that whatever progress was made in the surgery of the nervous system before 1860 we owe entirely to the surgeons, as the science of neurology up to this period was only in its infancy.

There is no more fascinating history than that of the surgery of the brain. It appears that trephining was practised by neolithic men, who inhabited Europe several thousand years ago.[1] They were supposed to have trephined principally for epilepsy and insanity. Most of the operations were performed on infants, and it is noteworthy that the majority of the holes found in the trephined skulls of that period were situated in the parietal region and over the motor area.

In the time of Hippocrates, 400 B. C., trephining was done mostly for injuries. From that period to modern times trephining passed through many epochs, sometimes falling into total disrepute, while in other periods it was the fashion to trephine the head at the slightest provocation. Thus, in 1664, the following curious certificate was written: "I, the underwritten, Philip, Count Nassau, hereby declare and testify that Mr. Henry Chadborn did trepan me in the skull twenty-seven times, and after that did cure me well and soundly." One wonders how the much-suffering Philip Count Nassau managed to survive these numerous operations in that day of no ether and sepsis.

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[1] The historical data were obtained from that admirable work of John Fletcher Horne on *Trephining in Its Ancient and Modern Aspects.* London, 1894, John Bale & Sons.

Injuries of the head were recognized, and with the exception of antisepsis, were treated with just as much skill centuries ago as at present, the more frequent occurrence of racial wars with the use of battering instruments making fractures of the skull common. The teaching of Hippocrates, that in all wounds an incision of the scalp should be made and the bone exposed can certainly not be improved upon. He even advised that resections of the bone should not be made on the temples nor above the temporal artery, for convulsions on the opposite side would be set up.

It is hardly necessary to trace this interesting history of the surgery of injuries in more detail, yet a word concerning the work of Baron Larrey, Napoleon's eminent general surgeon, would not be out of place.[2] This physician insisted upon the necessity of enlarging, by incision and drainage, all punctured wounds, trephined for hæmorrhage of the middle meningeal artery, besides for depressed and comminuted fractures, or any other condition that caused depression of the skull, and recognized the value of drainage in preventing pressure in intracerebral hæmorrhage. Baron Larrey's knowledge of the symptoms of cerebral injuries was in advance of his time. He recognized that injuries of the cortex would impair the intellect, and more important still he knew the value of relieving brain pressure by trephining.

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[2] *Johns Hopkins Medical Bulletin*, July, 1906. The achievements of Baron Larrey have been most interestingly written upon by J. Chalmers Da Costa.

One who sees many instances of cerebral injury cannot but be amazed at the remarkable resistance of the brain to septic conditions and injury. Consider, for instance, the frequent operations practised by the ancients, especially by the socalled trepanists in the seventeenth century, as in the case previously mentioned in which the skull was trepanned twenty-seven times. In the coal regions of this State, in depressed fractures of the skull, coal dust nearly always infiltrates into the wound, and yet sepsis is very uncommon. In the celebrated " crowbar " case the man lived for several years. In our modern wars, bullet wounds of the head sometimes are not followed by pus. A remarkable instance was told me by my friend, Colonel W. W. Wotherspoon, U. S. Army. After a skirmish in the Philippine Islands, he came across a young native lying unconscious in a trench. The boy's head had been laid open by a bolo, the knife having split the bone deflecting it to one side and exposing considerable brain substance. He was removed to an army hospital, the bone replaced and in a short time the boy was seen hopping around and, except for a slight hemiparesis, was in excellent condition.

Since the work of Broca in 1861 and the suggestions of Hughlings Jackson in 1864, soon followed by the work of Fritsch and Hitzig, and of Ferrier, between 1870 and 1875, our views of localization have made great strides, and it is because of this that nervous surgery has reached its present high position. Our views of the pathology of the brain and cord have made equal advances, and it is the combined knowledge of the physiology and pathology of the nervous system which enables us to-day to say when and where to operate or not to operate.

It was not until 1884 that modern views of localization were put to actual test in a case of brain tumor, although previous to this time focal operations had been done for traumatic and other conditions. Durante deserves credit for having done the first successful operation for brain tumor. In May, 1884, he removed a fibrosarcoma from the frontal lobe, the patient, according to report, living three months after the operation and being in good condition. In this country, Hirschfelder and Morse, of San Francisco, were the first to operate for brain tumor, an attempt being made to remove a glioma from the motor area on February 15, 1886, this being the fifth tumor operation recorded.

It might not be out of place to refer briefly to those early surgeons and neurologists whose work made possible the present position of nervous surgery. Among the former stand out prominently the work of Macewen, Horsley, Bergmann, and Krause of Europe, and of Weir, Park, Roberts, Keen, and Hearn of this country. Of the neurologists besides those already mentioned, the achievements of Schäfer, Munk, Luciani, Mott, and later of Flechsig, and of Weir Mitchell, Seguin, Mills, Putnam, Dana, and Starr of this country will ever be a source of inspiration to the students in neurology.

In this country great activity circled around the meeting of the Congress of American Physicians and Surgeons in 1888, at which time cerebral localization and brain surgery were much discussed. Charles K. Mills, of Philadelphia; Victor Horsley and David Ferrier, of England; Park, of Buffalo; Starr, and Weir, of New York, and Keen, of Philadelphia, taking a leading part in the discussion. It was at this time that Dr. Mills advocated the view of separate motor and sensory localization, and placed the motor centres largely in front of the central fissure, being one of the first, if not the first, neurologist to advance such views. He was the first to subdivide the sensory areas into distinct centres, views which now, eighteen years after, are first becoming generally admitted. It is largely to the work and enthusiasm of this neurologist that we owe the present high position of American neurological surgery.

It is curious that it should not have occurred sooner to surgeons that large cranial openings were of advantage. It was not until 1889 that the osteoplastic method of resection was first employed by Wagner. It is no wonder that not only were tumors not removed, but also not discovered through the small trephine openings practiced before this time. To-day cranial openings exposing one third of the lateral surface of a hemisphere are made with ease and with practically no danger to the patient.

Although in the majority of instances the removal of a brain tumor is not accomplished, this fact should not deter surgeons from operating, for an exploratory operation is at least as much indicated on the skull as on the abdomen. Such an operation has a double advantage in that it relieves intracranial pressure, thereby not only ameliorating the immediate symptoms, but also relieving headache and often preventing blindness by lessening the severity of the choked discs. In fact, too much emphasis cannot be placed upon the advantages of an early exploratory, or so called palliative operation. It has always seemed to me that in these palliative operations which are presumably done for the relief of intracranial pressure, very little if any cerebrospinal fluid escapes. If this is so, whatever benefit arises from such an operation is not due so much to the escape of fluid, but it may be, as Horsley has long thought, to the interference with the nutrition of the growth or because of mechanical reasons. A tumor growing in any portion of the cranial cavity will necessarily increase the intracranial tension, this probably being first manifested at the point of growth, and as the tumor grows, in all parts of the brain. Because of this, the dura is stretched causing pain, and the optic chiasm is compressed, this resulting in choked disc. In an operation when the dura is incised, the brain bulges because of the increased pressure, and whatever benefit results is due to relief of intracranial tension by the opening made, and not by escape of cerebrospinal fluid. One can readily forgive the ancients for thinking that it was because of the escape of noxious fumes that benefit resulted. The value of palliative operations has long been recognized, although in a crude manner. Celsus, writing in 30 A. D., says he saw a "melancholy man wounded in the head with a sword, his brain pan broken; so long as the wound was open he was well, but when his wound was healed his dotage returned again. Guianerius cured a nobleman in Savoy by boring alone, leaving the hole open a month together, by means of which, after two years of melancholy and madness, he was delivered." Since the introduction of modern surgery palliative operations have, of course, been frequently done, but it seems curious that the latest advanced procedure should be boring the skull for diagnostic purposes, steel or platinum-iridium needles being used for the subsequent exploratory puncture. These have a cavity and groove at their edge and along their longitudinal axis so that tissue may lodge. By this means Pfeiffer[3] was able to recognize by microscopical examination of the delivered tissue the presence of tumor in ten patients out of the twenty which were examined.

Surgeons have attempted to remove tumors from almost every portion of the brain. Cerebellar growths, the removal of which only a few years ago was hardly thought of are now occasionally successfully attacked. In the recent Toronto meeting of the British Medical Association, Sir Victor Horsley stated that he had operated for pituitary lesions ten times. The extent and growth of cranial surgery can perhaps be estimated by quoting the statistics kindly furnished me by Dr. C. H. Frazier, of the work done by him in the last five years in the hospital of the university of Pennsylvania. Cerebral operations were performed thirty-eight times, twenty-two of these being done for tumor, and sixteen for epilepsy, and the cerebellum was operated on sixteen times.

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[3] Pfeiffer, *Neurologisches Centralblatt.*, xxv, No. 13, page 639, July 1, 1906.

It is only proper that some reference should be made to the work done by surgeons throughout this great State, work which although unrecorded, is worthy of being classed with the best. Your honored chairman, Dr. W. L. Estes, performed twenty-nine brain operations before 1895. Similar work has been done no doubt by other surgeons in every part of the State.

I shall not devote much time to the consideration of technique, for that properly concerns the surgeon. It seems, however, always advisable that both the surgeon and the neurologist should agree upon the method of operation, and that the neurologist should be present to give whatever aid he can, especially in regard to interference with the brain tissue itself. Needless handling is to be deplored, as is also puncturing the brain with anything but a sharp needle. Many operations are made fatal, not by the opening itself, but by the subsequent treatment. Whatever the instrument used for opening the skull, whether it be a dental engine, chisel and mallet, or a De Vilbis saw, the object is to open the skull quickly. I have seen the skull opened in little more than three minutes by means of a dental engine. Contrary to the opinion of some English surgeons, rapid cranial openings are of decided advantage, for they not only shorten the time for the anæsthesia, but the brain is subjected to less jarring, and again, why lengthen any operation when it can be done just as safely in shorter time.

So far my remarks have been confined only to the surgery of injuries and tumors of the brain. It has always been a source of chagrin to the neurologist that so little can be done to cure cerebral hæmorrhage. Dural and cortical hæmorrhages should, of course, be operated on, but it is hardly justifiable to cut into a brain in search of a subcortical hæmorrhage. Even should the hæmorrhage be found, the actual destruction of the brain substance would counterbalance whatever questionable benefit would arise from the removal of a clot. Operations upon infants for the removal of dural or subdural hæmorrhages, as advised by Cushing, I would strongly indorse.

There is no more discouraging history than that of the surgery of epilepsy. Several thousand years ago, because of ignorance and superstition, the brain was exposed so as to allow the "demon in possession" to escape, as it was supposed that this caused the affection. Epilepsy was thought to be a sacred disease, and trephining was done as a sacred rite. Even at present exact knowledge of the disease is still wanting, and it is because of the various confusing theories advanced by as many different individuals that so many diverse operations have been practiced for the relief of this condition. To quote from Da Costa's paper on the *Surgical Treatment of Epilepsy:* "Various surgical methods of treatment have been recommended, including ovariotomy, clitorectomy, circumcision, nerve stretching and nerve section, orchidectomy, the removal of irritating scars and of painful cicatrices, blistering, cauterization, the use of seton, operations upon the ocular muscles to correct defects, the ligation of the vertebral arteries, the excision of the surgical sympathetic ganglion, and trephining of the skull." To this can be added various abdominal operations.

No one questions the advisability of operating in a case of epilepsy which is due to a definite cause, as an injury, hæmorrhage, or tumor, but to operate in cases of idiopathic epilepsy which have no discoverable focal basis either in the brain or in any other portion of the body, is to my mind a questionable procedure. In the first place, idiopathic epilepsy is an organic disease and is dependent upon structural developmental faults. These imperfections are not located in any one portion of the brain, as the motor area, but probably involve all parts. The epileptic convulsion is only the result of this maldevelopment; that is, only a symptom of a general disease. How futile, then, any focal brain operation!

Extracranial operations have been done and are still practised on the theory that toxines and other reflex causes occasion epilepsy. The surgical correction of ocular defects and the removal of nasal polyps may be an advisable procedure, but to ascribe these and similar defects as the causes of epilepsy is the veriest nonsense. Toxines unquestionably enter into the ætiology of the convulsion as a symptom; that is, given a structurally defective brain, it is necessary for a foreign element, perhaps a toxine, to bring on the convulsion; but there is no reason to believe that toxines themselves are the ultimate causes of epilepsy.

Much that has been said about the surgery of epilepsy can be equally applied to the surgery of idiocy, imbecility, and the various insane conditions. Fashions in surgery as in dress, have a habit of repeating themselves. As in epilepsy, the ancients believed that insanity and delirium were sacred diseases, or were due to the possession of the body and soul by demons and evil spirits, hence they opened heads to allow these to escape. Some modern surgeons open heads in insanity, idiocy, or imbecility to allow the brain to grow or to expand, believing that the growth or the function of the brain has been inhibited and that the opening of the skull will allow it to regain its proper size and function. The difference after all is not so great between the superstition of the ancients and the reasoning of some modern physicians.

The fault here, as in epilepsy, lies in the fact that symptoms are taken for the disease itself. Most insane conditions have an organic and, usually, an embryological basis, a view only recently expressed by Dr. Mills in the Toronto meeting of the British Medical Association, and one with which I am in accord. Idiocy, imbecility, and such insane conditions as dementia præcox, epileptic insanities, and paranoia are unquestionably due to developmental faults. With Dr. J. Chalmers Da Costa, I believe that operations for such cases do about as much good as attacking a thermometer for the purpose of regulating the existing temperature!

Some physicians believe in the advisability of operation for hypochondriacal delusions and for hallucinations. Delusions and hallucinations are only symptoms and are liable to change, as in one of Dr. Da Costa's cases, in which the patient believed that he had a kitten in his viscus. An emetic was applied and a cat was shown him in the bucket. The patient seemed satisfied, but within an hour he stated that he had something else in his stomach,—an eight day clock!

Not much advance has been made in the surgical treatment of hydrocephalus. It is difficult to understand how even permanent drainage of the lateral ventricles could cure the condition, presuming that such surgical procedure is successful. The secretion of the cerebrospinal fluid is probably the result of over action of the choroid plexus, and drainage will not stop further secretion. After the ventricles are thoroughly dilated it is difficult to understand what benefit could result from relieving them of the fluid which they must have. Altogether, I am of the opinion that operation on hydrocephalic conditions are contraindicated and that we have been laying too much stress upon the possible benefits of the relief of intracranial pressure, by the depletion of cerebrospinal fluid.

SURGERY OF THE SPINAL CORD.

It has only been in comparatively recent years that the spinal cord has been considered a fruitful field for surgical procedure, the first successful operation being performed by Horsley in a case with Gowers, of an intradural fibrosarcoma of the upper thoracic region. The growth was removed, June 9, 1887. Ten years elapsed before the second successful operation was performed by Freeman in a case with Eskridge, in 1887, in this country. Since then spinal operations have become more frequent. There is every reason that such operations should be successful, for our knowledge of spinal localization is fairly exact, at least more so than that of the brain.

Spinal tumors, if not multiple, are as a rule easy to enucleate and are benign; one of the most successful operations I know of was performed by Edward Martin in a case of J. H. Musser and W. G. Spiller, an intradural cyst being found and removed.

One of the most difficult questions to decide is the advisability of operations in cases of fracture and dislocation, and spinal injury. As a rule, if there is a fracture and dislocation the spinal cord has been severely crushed, and even though pressure is removed the cord will not be benefited. Of course, it is good surgery to reduce a fracture and replace a dislocation, but to my mind the dangers of sepsis and the fact that no regeneration can occur in the spinal cord, contraindicate surgical interference, at least in the more severe cases. In spinal injuries in which there is no fracture or dislocation, hæmorrhage may occur either in the centre of the cord or in minute areas throughout its whole extent. Here an operation would be of no value. In the first place, it is impossible to get at the hæmorrhage, and again, if the spinal cord is left alone, if any recovery is possible outside interference will be only detrimental.

It has been thought by some surgeons that in cases of complete severance of the spinal cord, suturing of the ends may cause regeneration and a consequent return of power. This, to neurologists who are aware of the fact that regeneration does not occur in the spinal cord, seems an error. Experiments upon lower animals have proved that this is impossible. After a spinal cord is completely severed it would be rather difficult to approximate crushed ends. Even if this were successfully done, so much tissue would be destroyed on either side that regeneration, were it possible, could hardly proceed.

When lumbar puncture was first introduced it was hailed with much enthusiasm, but, unfortunately, we have learned that it is a dangerous procedure and of not much benefit. The reason is that as in the cranial palliative operations, the actual removal of the cerebrospinal fluid, while it may temporarily relieve pressure, is of no value in either curing or ameliorating the original cause of this excess of tension. The danger of the operation lies in the sudden disturbance of pressure, and it is possible that the sudden deaths recorded as the result of this operation may be due to the disturbance of circulation in the bulb. Lumbar puncture for diagnostic purposes is, however, sometimes justifiable.

The value of the Röntgen ray for diagnostic purposes in nervous affections has also proved disappointing. Many claims have been made for it, but so far it would be unwise to risk a diagnosis upon its indications. It is only a question of time, however, when the technique of x-ray work will be so far advanced that great aid will be given to focal diagnosis. In the recent Boston meeting of the American Medical Association, Dr. Edward Angell, of Rochester, New York, exhibited a new stereoscopic apparatus by means of which the outlines of cranial lesions could be most clearly seen.

SURGERY OF PERIPHERAL NERVES AND MUSCLES.

Perhaps more progress has been made in the surgical treatment of peripheral palsies during the last decade than in the other branches of nervous surgery. Barely eleven years have elapsed since Ballance and Stewart, of England, joined the facial nerve to the spinal accessory for the relief of facial palsy, this being the first instance of nerve grafting performed on man. Since then, nerve anastomosis has been done in cases of anterior poliomyelitis, hemiplegia, for the relief of athetosis, and in many instances of palsies of peripheral nerves.

That regeneration of peripheral nerves is possible was known in 1795 by Cruikshank, who was apparently the first to recognize that severed nerves could grow together, although this honor has been usually given to Flourens. It seems curious that these experiments should have been so long forgotten, and that its principles should not have been applied until just one hundred years after their discovery.

It was not until the work of S. Weir Mitchell, Geo. R. Morehouse, and W. W. Keen, appeared in 1864 that the surgery of peripheral nerves was placed upon a fundamental basis. Later, Weir Mitchell published a separate volume on the peripheral nerves. Many of the descriptions of nerve palsies found in this admirable book can even at this date not be improved upon. His description of the first case of sympathetic paralysis ever recorded is classic.

Neurectomy and nerve stretching have been done for many years for the relief of neuralgias, and for neuritis, and have even been done for the relief of the contractures in cerebral and spinal palsies. Happily, nerve stretching is largely being abandoned as a therapeutical measure. In a disease like sciatica it is difficult to understand how stretching the nerve will relieve the pain any more than stretching the fifth nerve will relieve tic douloureux; as in the latter disease it might be a justifiable procedure to cut the sensory roots in the spinal cord for the relief of sciatica.

It is not my purpose to dwell at any length on the principles of nerve regeneration. Whether the central or the peripheral theory is accepted, the important factor is that regeneration will take place between the cut ends of a peripheral nerve unless mechanical means are taken to prevent such union. I will also leave it for the surgeon to decide whether end to end anastomosis is preferable to end to side, or other methods. I shall only speak of the therapeutical application of nerve transplantation.

In facial paralysis the result of an ordinary cold in which no improvement has taken place for six months, in palsies, the result of middle ear disease, or in cases of accidental severance, suturing of the ends in the latter case or anastomosis with the hypoglossal or the spinal accessory nerve is clearly indicated. This operation should also be considered in spasmodic facial tic. Peckham, of Providence, was the first to do nerve anastomosis in anterior poliomyelitis, in 1899. Independently of this, Spiller, in 1902, recommended this operation, it being successfully performed by J. K. Young. Later Spiller applied the principles of nerve anastomosis in hemiplegia and in a case of athetosis, in which brilliant results were obtained by Frazier.

In considering the surgical means to be employed for the cure of peripheral palsies, tendon transplantation must not be overlooked. As a therapeutical measure it was first introduced in 1881 by Nicoladoni. Parrish, of New York, deserves credit for having been the first American to perform this operation, in 1892. Great advances were made by Goldthwait, of Boston, who, in 1895, was the first to do muscle transplantation, and by Lange, of Munich, who first advocated suturing tendons to the periosteum.

In choosing between the two operations, nerve transplantation is obviously more advantageous, because by this means function would be restored to the paralyzed muscles. As in all new operations much discouragement will at first result, because in almost all cases of palsy such therapeutical measures will be considered, whereas in only certain selected cases can good results be expected.

In an address on the surgery of the nervous system reference must be made to the surgery of tic douloureux. This malady, which, according to Cushing, was accurately described by Avicenna about nine hundred years ago, has baffled successful treatment even up to the present. This is because even now its pathology is not understood. We do know that by cutting all connection between the peripheral ends of the trigeminus and the pons, either by extirpating the ganglion of Gasser or by cutting its sensory root, the pain will cease.

Many operations have been proposed and done for the relief of this condition. The seventh nerve has been cut, the peripheral branches have been excised, or stretched, but it was not until 1890 that Macewen and Horsley, independently of each other, attempted to divide the trigemineal nerve intercranially. Rose, of England, soon after first attempted to remove the Gasserian ganglion. This operation, which at first was nearly always fatal, is now performed without much loss of blood and with success. Only recently (1904) Cushing reported twenty cases, and he writes me that since then he has had a second twenty with much better results.

After the medical treatment of tic douloureux has proved unavailing, it is a safe rule to first consider such peripheral operations as neurectomy, except in the more severe cases in which it is advisable to perform a major operation at once. Recently, Murphy, of Chicago, has recommended exposure of the peripheral nerves with injection of osmic acid into its sheath.

Of the more radical operations, mention should be made of the suggestions of Van Gehuchten, that the peripheral branches should be torn out, believing that this has a reactionary effect on the nerve cells in the ganglion, not only destroying these but also causing degeneration of its bulbospinal root. This operation was originally suggested by Blum in 1881. Abbe advised intracranial section of the second and third divisions of the trigemineal nerves. None of these operations, however, have been sufficiently done to pronounce judgment. The operation of choice is either removing the ganglion of Gasser, or cutting its sensory root. On physiological grounds, the latter operation is to be preferred, providing, of course, that regeneration does not occur, a fact which has now been established. This operation was first suggested by Ferrier, in 1890, in the discussion of Rose's operation, and was first performed by Horsley, although the operation was not done with this definite object in view. Spiller, in 1898, again suggested it, this operation being then undertaken by Frazier in 1901, it being the first time that the sensory root was successfully cut, the pain ceasing entirely.

In conclusion, when we consider that most of the advances in neurological surgery have been made in the last twenty years, and not a few of these in the last decade, we cannot but feel that there is much hope for still greater advances in the near future. As regards the surgery of the brain, these advances will probably come in the first place through our slowly increasing knowledge of subdivisional cortical localization and through improvements in the methods of opening and exploring the cranial cavity. What has been recently done in nerve anastomosis forecasts more brilliant achievements.

In closing, I cannot refrain from referring to the great influence for good which such combined meetings as those of the surgical and neurological sections of the American Medical Association, and the sections of this society have exercised by bringing together men in different but correlated lines of work.

2030 Chestnut Street.

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THE SUBDIVISION OF THE REPRESENTATION OF CUTANEOUS AND MUSCULAR SENSIBILITY AND OF STEREOGNOSIS IN THE CEREBRAL CORTEX.*

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The main object of the present contribution is to present the following propositions: (1) that the cortical representation of cutaneous and muscular sensibiltiy is independent of motor representation, that it surrounds the motor zone, and that it is subdivided into a mosaic of centres, each centre or group of centres being anatomically and functionally correlated to a motor centre or centres; (2) that every muscle or group of muscles producing a movement or movements which are represented by separate centres in the cortex is topographically related to a segment of the skin which has also a definite cortical centre, this centre being correlated anatomically and functionally with the motor centre; (3) that stereognostic representation like that of cutaneous and muscular sensibility and of movements has also its independent cortical area, and is subdivided after the manner of the motor and sensory areas.

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*Read before the American Neurological Association, Boston, Mass., June 4, 1906.

It will be seen that according to the above propositions, the representation of cutaneous and muscular sensibility and stereognosis is not only for the face, arm, trunk, and leg, but for subdivisions of these parts.

The acceptance of the view that the motor zone is entirely cephalad of the central fissure gives a position of advantage in discussing the question both of separate motor and sensory representation and of the subdivisions of the latter. Those who believe that the cortical motor and sensory areas are identical have until recently been in the majority, but the weight of opinion is now in favor of the separatists.

The writers accept without qualification the view that the motor area is cephalad of the central fissure. One of them (Dr. Mills) has been a consistent advocate of separate motor and sensory representation. As early as 1888 he attributed sensory functions to the parietal lobe, and also in several publications (1888, 1895 and 1904)[1] expressed the view that the cortical sensory area was subdivided into centres in a manner similar to the subdivision of the motor area.

The following quotation from the paper published in 1904 gives his views as then expressed:

"It is probable that the areas of representation of cutaneous and muscular sensibility are subdivided in a manner analogous to the subdivision of the motor area, in other words, for the face, head, neck, upper extremity, trunk, lower extremity and other parts of the body, and that even some of these areas contain subareas and centres. As indicated diagrammatically, the cutaneomuscular sensory area encircles on all sides the great motor area on the lateral aspect of the hemispheres, occupying the postcentral convolution and the anterior extremities of the superior and inferior parietal gyri or lobules, and on the mesal aspect the more posterior and inferior portions of the paracentral and frontomarginal gyri, and the contiguous parts of the precuneus and gyrus fornicatus. The sensory areas for the face, head, neck, upper extremity, trunk, lower extremity, etc., occupy respectively areas adjoining the subdivisions of the motor cortex representing these parts of the body; in other words, that the areas and subareas of representation of the movements of the larynx, jaws and face should be toward the lowermost portion of the postcentral and postparietal convolutions, the areas for the head and neck, upper extremity, and trunk coming next in order from below upward, while on the lateral edge of the hemisphere and on its mesal aspect are centres for the lower extremity, anus, genitalia, and viscera. Clinicopathologic, experimental and psychologic facts can be adduced in support of these localizations. Studies in hypnosis, and in hysteria and of the sensory aura of Jacksonian epilepsy point in the same direction. A considerable number of cases of limited cortical or of subcortical lesion have been recorded in which the anesthesia has been circumscribed to a limb or the face or trunk, or to special portions of these larger divisions of the body. The well-known experiments of Horsley and Schäfer also showed that circumscribed areas of anesthesia sometimes resulted from experimental lesions of the limbic lobe. Grünbaum and Sherrington, in their experiments on the higher anthropoids, demonstrated that certain segments of the non-excitable postcentral convolutions were distinctly related to particular subdivisions of the precentral motor cortex. Faradization of portions of the cortex of the postcentral convolution, while not calling out motor response, facilitated this response in portions of the precentral convolution at the same horizontal level. This is a fact pregnant with suggestion."

At a meeting of the American Neurological Association, held in St. Louis, September 15, 16, and 17, 1904, Dr. Mills presented a paper on the location and subdivision of the cortical areas of cutaneous and muscular sensibility. This paper, however, has never been published, and some of its data have been incorporated in the present article.

The view that an area jointly sensory and motor is subdivided into centres of special representation for different portions of the body (Dana, Starr, Horsley and others) must not be confounded with that which holds in the first place, that the areas of representation of movements and of sensibility and of stereognosis are separate: and secondly, that these are subdivided into subareas and centres. It is the latter doctrine which is taught in this paper.

The evidences which favor the subdivision of the sensorial and stereognostic cortex are chiefly clinical cases with or without necropsy in which this subdivision is indicated. The experimental and histological data with regard to cortical motor and sensory localization up to the present time, chiefly have reference to the position and extent of the zones of representation and not to their subdivision. The clinical and clinicopathological cases are of two kinds: those in which symptoms indicate both motor and sensory subdivisional representation and those in which sensory representation alone is illustrated.

We shall not recall here those cases now well known in neurological literature in which both motor and sensory subdivisional localization are indicated, that is, cases in which both loss of power and impairment of sensation were confined to a leg or an arm, or the face or to subdivisions of these portions of the body. These cases are scattered through the literature of the last twenty-five years, and most of them are to be found in the elaborate digests of cases published by such writers as Starr[2], Dana[3] and Verger[4].

Dana, in discussing the cases collected by him, said that the anesthesia was always circumscribed to the face or limbs or to a special part of the body; it was never complete hemianesthesia when the lesion was cortical. The sensory centres for various parts of the body, as for the face, arm and leg, were however according to him, commonly identical with the motor centres for those parts, but were larger and more diffused. Starr held to a similar view.

We shall now call attention to a series of cases, comparatively few but important, which seem to indicate a large degree of subdivision of the areas of representation of cutaneous and of muscular sensibility and of stereognosis. In the presentation of these cases those first given point only to the subdivision of the cortical areas of representation of cutaneous and muscular sensibility. The later cases indicate that the stereognostic zone is similarly subdivided. In considering the first cases (Knapp, Darkschewitsch, Madden and Starr and McCosh) it must be remembered that they were recorded before the subject of stereognostic conception and astereognosis had been considered in neurological literature. In some of them, therefore, it is probable that astereognosis, as well as impairment of cutaneous and of muscular sensibility, was present.

A case recorded by Knapp[5] in 1891 is confirmative of the subdivisional sensory representation in the cortex and might also be regarded as evidence in favor of the separate cortical representation of movements and of sensation, as the lesion was presumably behind the central fissure. The most interesting fact in the present connection is that the loss of cutaneous sensibility involved only the hand and forearm. This case was one of traumatic brachial monoplegia with limited loss of sensation. The patient had received a wound on the head during the Civil War and was operated on about thirty years afterwards. During the operation the surgeon injured the cerebral cortex in what was supposed to be the middle portion of the left postcentral convolution, the lesion being recorded as about one centimeter long and about three millimeters in depth. The results of this surgically produced lesion were disorders of sensation, which continued while the patient remained under observation. They are recorded as having been present at the end of the year. Tactile sensibility was much diminished in the right upper extremity; the patient had no consciousness of the position occupied by the extremity; muscular sensibility was lost in it. The sensations of pressure and weight were also lost, but pain and temperature senses appeared to be normal. The loss of cutaneous sensibility extended only to the elbow.

Darkschewitsch[6] recorded a case in which general sensibility was entirely diminished in all its forms in the right upper extremity, and especially in its periphery. Sensibility was unaffected in the right leg and right side of the face, and these parts also presented no affection of motility. At the necropsy a solitary tubercle was found in the white substance of the left cerebral hemisphere, subjacent to the cortex of the middle portion of the postcentral gyre. It will be seen that this lesion corresponded closely to what we regard as the subarea of cutaneous and muscular sensibility for the right upper extremity.

Madden[7] recorded a case of cortico-subcortical tumor with apoplectiform attacks preceded by a sensation of pressure, with incoördination of the arm and forearm of the left side followed by paralysis of the leg and arm of the same side. Examination showed that on the left hand tactile sensation was much altered and that it was completely lost in the ring finger. The muscular sense was also lost, and at times there was marked ataxia. The disorders of sensibility became more and more apparent as time advanced. At the necropsy a prominence, thinning of the dura and depression of the cranium were found on the left side at a point half an inch to the left of the longitudinal sinus in the postcentral gyre. Symmetrically situated on the opposite side was a smaller prominence. Underneath this prominence was found a cystic tumor which involved the postcentral, the superior parietal, supra Sylvian, and the inferior portion of the angular and occipital convolutions.

Starr and McCosh[8] reported a case in which, for the relief of spasmodic attacks, trephining was performed and a small angioma removed. The operation was followed by loss of muscular sense in the opposite hand and forearm without any disturbance of other sensations or of movement. There was also marked ataxia in this limb. The place of operation corresponded to a spot about the junction of the superior and inferior parietal convolutions, and distinctly posterior to the postcentral convolution.

In a case reported by Klien[9] of trauma of the left side of the skull, in which the injury was supposed to have involved both the precentral and postcentral convolutions, there was a resultant paresis of the right side of the body. Sensation for light touch was diminished over the whole right side, but touch applied a little more deeply was disturbed only in the right arm. This was especially so in an irregular area over the radial side of the right hand and forearm. The pain sense was disturbed over this same area, but temperature sense was somewhat altered over the whole right side. The sense of position and stereognostic conception were disturbed in the whole arm, and there was considerable ataxia of this arm.

Bonhoeffer[10] in a study on disturbances of sensation due to cortical lesions, reported a case (case 4) in which, because of a depression of the right parietal bone, paresis of the left arm, and especially of the hand was present. Sensation for pain was diminished over the whole left side, but especially so in the upper limb. The sense of touch, of localization, of motion, and of position, and stereognostic conception were impaired over the whole hand, but especially so in the third, fourth and fifth fingers.

Fischer[11] reported a man of twenty-three years, who because of numerous cysts in the cerebral cortex, had attacks of Jacksonian convulsions, which always began with a feeling of numbness in the fourth and fifth fingers of the left hand. The patient was operated upon in the right motor area and a cyst due to a cysticercus was excised. The patient improved, but subsequently the Jacksonian attacks reappeared.

When examined by Fischer sensation for touch and pain was lost in the three ulnar fingers and in the ulnar side of the hand both in the palmar and extensor surface up to the wrist. From here to the elbow there was hypesthesia for touch and pain. This sensory disturbance faded imperceptibly into the surrounding parts. The sense of localization, of movement, and of position, and stereognostic conception were lost only in the fourth and fifth fingers. Power was little if at all disturbed in these parts. Several days after the examination giving the above results, a complete hemianesthesia for all forms of sensation was obtained, and other symptoms regarded as hysterical were also found. Suggestive treatment removed the sensory disturbance, and because of this the ulnar disturbance of sensation was regarded by the author as hysterical in origin.

It is probable that the disturbance of stereognostic conception and of other forms of sensation in the ulnar side of the hand was of organic origin, and that the subsequent hemianesthesia was due to a superimposed hysterical condition.

Kramer[12] in 1896, in a paper on "Tastlähmung" or touch paralysis, reported a number of cases of localized disturbances of sensation due to cortical lesions. Among these, in one case (case 6), a man of forty-two, after a sudden attack of dizziness, developed weakness of the left upper limb with increased reflexes. Sensation in the left hand for touch, for pain and for temperature was normal. On the third, fourth and fifth fingers, however, there were very mild disturbances of sensation of touch as well as of the sense of localization. The sense of movement was disturbed in the second and third fingers.

In a paper on the sensory disturbances occurring in hemiplegia Sandberg[13] reported a number of cases of localized disturbance of sensation. In one case (case 1) a man of forty-five, with a history of Jacksonian convulsions limited to the left side of the body, and beginning in the left face, when seen some years after the spasms were noted, showed no motor involvement anywhere. The reflexes were normal. Touch-sensation was somewhat disturbed, especially in vola manus, as well as in extensor surface of the hand. Sensation for temperature and for pain was normal. The pressure sense was disturbed in the whole hand.

The sense of movement as well as the sense of localization and stereognostic conception were disturbed somewhat in the whole left hand and especially so in the third, fourth and fifth fingers. Sensation in the right side was normal.

Of the nine (9) cases thus briefly abstracted, only in those in which necropsies were had or in which the operative procedure indicated clearly the site of the lesion, can it be said with positiveness that the lesion was entirely behind the central fissure. Apparently it was behind in at least five of the cases. In all the lesions were in part at least behind the fissure.

The cases are particularly interesting as indicating the limitations of anesthesia and astereognosis, not only to the upper extremity, but to limited portions of this limb, and especially to the hand and certain of the fingers.

The impairments of sensation present were in several instances more or less dissociated, certain forms of impairment being present and others not, or different forms showing themselves in different parts.

It is interesting to note that in four of the nine cases the impairment and disturbance of sensation were greater in the fingers towards the ulnar side of the hand, this showing itself sometimes in two and sometimes in three fingers.

The cases clearly indicate the subdivisional representation in the cortex, and most of them that this subdivisional representation is for definite portions of the extremity, as for certain fingers or for the ulnar or radial portions of the hand.

To these nine cases we shall add four studied by ourselves.

Case 1.—*Traumatism and syphilitic meningitis of the parietal lobe; Jacksonian epilepsy beginning in the fingers and hand; astereognosis in the left hand; tactile anesthesia and impairment of pain sense in the left upper and lower limbs; operation for removal of diseased mass from the parietal region; transient paralysis after operation with persistent sensory loss and astereognosis; objects recognized by the left thumb and forefinger, but not by the other fingers, which showed greater impairment of sensation and of co-ordination; second operation and further removal of diseased tissue; astereognosis, ataxia, and impairment of sensation now the same in all parts of the hand and fingers; necropsy showing widespread gummatous meningitis and an area of softening in the postcentral and inferior parietal convolutions, to a very slight extent invading the precentral convolution.*

This patient was a man of 41 years, who for four years had been under our observation either in the Polyclinic, the Philadelphia General, or the University Hospital. He had been at various times under the care of Dr. Wm. G. Spiller. He was not married and admitted a specific history. He had been a hard drinker and five years before he first came under our observation, while having a fight in a saloon, was thrown out into the middle of the street, and was taken to the Pennsylvania Hospital, where it was said that he had concussion of the brain. He was unconscious for about twenty-four hours. Four years after this, while again drinking, he fell backwards and struck the back of his head on a brick sidewalk. His head was cut in the parietal region a little to the left of the median line.

When he first came to the dispensary of the Polyclinic Hospital, April 29, 1902, examination showed the right side of the head to be more prominent in the parietal region. About 9 cm. (3.54 in.) above the attachment of the ear to the head was a depression in the bone about the size of a little finger. This area was very sensitive to pressure. Dullness was obtained over this part. The patient dated his headache from this accident, it being frontal and persisting night and day. He also had what he called sharp neuralgic pains. At times he vomited after eating and he also complained of nausea.

On Feb. 3, 1902, he had his first convulsive attack. This began with pain in the tips of the fingers of the left hand; then passed to the back of the hand, and according to his description, his fingers were twisted in every shape. The convulsion then crept up his arm, the patient experiencing pain in the top of the left shoulder, after which he lost consciousness, frothed at the mouth, but did not bite his tongue nor void urine. His arm was not weak after the attack.

When examined, station and gait were normal, the eyes both as to condition of the pupils and extra-ocular muscles were normal, and sensation was normal all over the body. The reflexes were also not altered except the right patellar jerk which was slightly increased. Even at this time he called a quarter a five cent piece in the left hand. Motor weakness was everywhere absent.

From this time until his admission to the Polyclinic Hospital, April 29, 1902, he had several convulsions, all of which were similar in character to the first, except that they became longer. He would at times complain of pain as if an electric shock would start in his hand and travel from the finger tips to his forearm and arm, and his fingers would feel cold and numb after the attacks. On his admission to the hospital examinations showed that the grip of the left hand was not as strong as that of the right, and that he could not recognize any object placed in the left hand.

Five days before the operation was performed upon him examination by Dr. Spiller showed complete loss of stereognostic conception in the left hand and complete tactile anesthesia and impairment of the sense of pain in the left upper limb. Sensation for touch and pain was also diminished in the left lower limb. The reflexes in the left upper and lower limbs were increased, but not markedly exaggerated. Ankle clonus and Babinski response were absent on this side. Sensation and the reflexes on the right side were normal. The sense of position in the left arm was lost and was impaired in the left leg. The eye examination did not show anything abnormal except a slight paleness of the left optic disc.

The examination of the patient, five days later, just before the operation, showed almost complete paralysis of the left upper limb, the patient being in a mild stupor, although able to answer questions. Sensation seemed about the same as in previous examinations.

Dr. John B. Roberts, under complete anesthesia, operated over the right parietal bone, and after dissecting up an oval flap he found a spot of carious bone about the size of half a dollar. This was easily removed by bone forceps. The dura was thick and was cut away, exposing a hard mass about the size of a pigeon's egg, with several small nodules attached. A part of this mass was brain substance.

The patient made a good recovery. Examination several days after the operation showed almost complete paralysis of the left upper limb and some paralysis of the left lower limb. Tactile sense was almost entirely lost in the left upper and lower limbs, pain and temperature senses were only slightly impaired in the left upper limb and preserved in the lower limb, the sense of position was lost in the fingers and toes on this side. The reflexes were exaggerated, but there was no Babinski response. About three weeks after the operation the man left the hospital. The paralysis in the left upper limb had almost completely disappeared, the return of voluntary power being remarkable in view of the complete paralysis which existed previous to the operation, and the fact that considerable brain tissue had been removed. Stereognostic conception was completely lost in the left hand and there was still some impairment of sensation in the left upper limb.

The miscroscopical examination of the specimen removed at the operation showed intense inflammation and round cell infiltration, suggesting syphilis and gumma of the cortex.

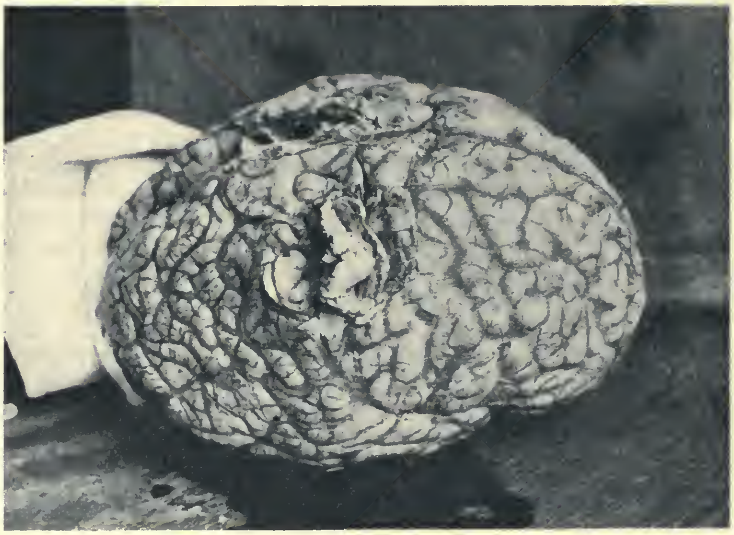
For six months after the operation the patient was well and he had no convulsions. He was completely free from headache, nausea and vomiting. He had, however, in the meantime resumed his previous alcoholic habits, and he had not been regular in his taking of potassium iodide, which was given to him after the operation. About six months after his operation he began again to complain of convulsions, and these were similar in character to those previously described. When examined, March 9, 1903, about nine months after the operation by Dr. Weisenburg, the patient complained of considerable persisting frontal headache, nausea and vomiting. His gait and station were good. The grip of the left hand was weak, this being the only sign of motor involvement. Sensation for touch and for pain was disturbed over the left upper limb, and somewhat in the left lower limb. The reflexes were generally increased, but were more so on the left side. He called attention to the fact that he was able to recognize objects placed between his left thumb and forefinger, but could not recognize any object in his other three fingers. Examination confirmed this fact. He stated that he had had this condition since his recovery from the operation. A further examination showed that the sense of position was lost entirely in the three ulnar fingers, but was preserved in the thumb and forefinger. Sensation for touch and for pain was also disturbed more in the ulnar three fingers and less so over the skin covering the thumb and forefinger and the radial side of the hand. In the finger to nose test considerable ataxia was apparent and this was also more marked when the ulnar fingers were directed to the nose.

He constantly complained of an agonizing pain in the left upper limb; in fact, he came for treatment principally because of this and his convulsions. He stated that his left upper extremity constantly felt as if it were being cut by a knife or as if he were having a convulsion. These pains existed for over two years, until the time of his death. Towards the latter end of his life he would also complain of pain in the lower limb and sometimes in his face, chest and abdomen, but mostly the pains persisted in the left upper limb. The convulsions would always start in the left upper limb. During these the pains would become excruciating. During most of the convulsions he would not lose consciousness.

Because of the increase of headache, nausea and vomiting, and the increasing number of convulsions, he was admitted to the University Hospital and was again operated upon, this time by Dr. C. H. Frazier, on Oct. 15, 1904. In the operation the incision followed the line of that made at the original operation. The scalp and dura seemed more or less fused together and the dura was adherent to the substance beneath. After the dura was opened, grayish tissue was found beneath and scraped away. The brain was much congested. Gummatous tissue was also scraped away from the inner side of the dura.

The patient was not much benefitted from the operation. The convulsions were just as marked and the pains just as severe. He had, however, some relief from his headache, nausea and vomiting.

Ophthalmoscopic examination at no time revealed anything but a slight paleness of the optic discs of both sides. The convulsions now, instead of always starting in the fingers of the left upper limb, would sometimes start in these and again in the abdominal muscles of the left side. The patient's condition grew gradually worse, the convulsions became more frequent and the pains of such intensity that he had to be kept constantly under the influence of narcotics and hypnotics. Examination just before his death showed some weakness of the left upper limb, but none of the face or of the lower limb. Sensation for touch, for pain, and for temperature was disturbed over the left upper limb and less so over the left lower limb.



Photograph of Brain of Case 1. Area of Softening Involving Part of the Upper Portion of the Postcentral Convolution and the Anterior and Superior Portions of the Inferior Parietal Convolution. A Little of the Precentral Convolution Was Also Involved.

Directly after the operation by Dr. Frazier it was noted that he was unable to recognize objects placed in his whole hand, whereas previous to the operation he could do this between his thumb and forefinger. The loss of the sense of position, the ataxia, and the impairment of sensation all seemed to be equally marked in all portions of the hand. The patient died about Jan. 1, 1906.

Necropsy was performed by one of us (Dr. Weisenburg). The scalp over the right side of the skull was adherent, especially over the site of the operations, and had to be dissected away. The right portion of the skull was exceedingly carious and was cut away without much effort. The bone and the dura were adherent almost over the whole surface of the brain, but especially so over the right side where it had to be dissected away. On removing the skull a thick gummatous meningitis was found, this being marked over both hemispheres, but especially so over the right hemisphere. Over the right parietal area it was at places almost a quarter of an inch thick. When removed, an area of softening was found over the right parietal lobe. This area, as indicated in the photograph, (Fig. 1) involved part of the upper portion of the postcentral convolution, a very little of the adjacent precentral convolution, and the anterior and superior portions of the inferior parietal convolution. The microscopical examination showed typical syphilitic exudation.

A careful examination of the spinal roots of the fourth, fifth, sixth, seventh and eighth cervical and first thoracic segments of either side showed no round cell infiltration. The roots were cut separately.

Case 2.—Parietal injury with unconsciousness; ataxia, loss of the sense of position, astereognosis, and impairment of the senses of touch, pain, temperature and spacing in the hand and upper extremity; later cutaneous sensibility, the senses of position and of movement, and stereognostic conception better in the thumb and forefinger and radial side of the hand and forearm than in the other fingers and ulnar side; touch and pain senses were more disturbed in the distal than in the proximal portions of the hand; pain sense more appreciated in the palmar than the dorsal surface.

The history of this patient was given by Dr. Spiller in the Feb., 1906, number of the JOURNAL OF NERVOUS AND MENTAL DISEASE, in a paper on separate sensory centres in the parietal lobe for the limbs. We will first give his account of the case, somewhat condensed. A man, 38 years old, was struck over the right parietal lobe with a club on July 21, 1904, was unconscious for one or two hours and was confined to his bed about ten days after receiving the blow. For two weeks he could not move the fingers of his left hand, either as the result of motor weakness or of sensory disturbance. The hand, which at first felt "dead," had been paresthetic since the injury. He had never had any objective or subjective disturbances of sensation in the left lower limb or face.

When examined on Dec. 11, 1905, the movements of the left upper limb were awkward, although it could be moved freely at all parts and was not weak. He was unable to button his coat with his left hand alone when his eyes were closed, because of incoördination of the fingers, but could button it promptly with his right hand. The sense of position was greatly impaired in the fingers of his left hand, and he was usually unable to tell which finger was moved or when a finger was bent backward or forward. Stereognostic conception was also greatly impaired in his left hand, and usually he was unable to name any object placed in this hand if he did not see it. Occasionally if the object fell from the hand he was unaware of the loss. Sensations of touch, pain and temperature were diminished in the left hand, but not to the same degree as were the sense of position and stereognosis. He occasionally answered correctly when his left hand was touched or pricked with a pin, but usually his answers were uncertain and in marked contrast to the positiveness with which he replied to sensory tests of the right hand. The sensation of touch was a little less acute in the left hand than in the left arm above the elbow. Pain sensation was less altered than tactile sensation, and temperature sensations for both cold and warmth were more nearly equal in the two upper extremities. The spacing sense was also greatly altered in the left hand, and he could not tell whether the thumb and first finger were near together or far apart. In the right hand he could tell whether the thumb and first finger were one or two inches apart. Sensations on the chest seemed to be the same on the two sides.

The left lower limb was not affected. His answers to sensory stimuli were always correct and prompt. He recognized at once any movement of the toes and the direction in which they were moved. The limb was not weak. The patellar reflexes were not prompt. Sensation in the left side of the face appeared to be entirely normal, and the right side of the body was not affected. Hemianopsia was not present.

The case, according to the writer of the paper, seemed to indicate that the sensory centre for the upper limb was distinct from those for the face and lower limb.

When this patient was examined by one of us (Dr. Weisenburg) on Feb. 19, 1906, he was able to recognize a knife between the thumb and forefinger of the left hand, but was not able to do so with the other fingers. He was also able to recognize properly a coin, as a quarter, but in a few minutes his ability to recognize objects placed between his thumb and forefinger was not so good. Sensation for touch and pain was better on the skin over the thumb and forefinger than over the ulnar part of the hand. This difference in sensation between the radial and ulnar portions of the limb was also apparent on the skin covering the forearm up to the elbow, this differentiation not being apparent above this point. It seemed as if touch and pain were better recognized as the radial side of the hand and forearm was approached.

The sense of position was lost in the three ulnar fingers, impaired in the forefinger, and about normal in the thumb. The same could be said of the sense of pressure, the sense of localization and the sense of movement; in other words, the sense of pressure, of localization, of position and of movement were all better recognized as the radial side of the hand was approached. These different perceptions were lost or almost entirely lost in the two ulnar fingers, appreciated in the middle finger, better appreciated in the forefinger, and almost normally appreciated in the thumb. Bone sense, or that sense which is obtained by touching the bony parts with a vibratory tuning fork, was similarly better recognized as the radial side of the hand was approached. Repeated subsequent examinations invariably demonstrated the above facts. The ability to recognize objects between the thumb and forefinger was sometimes quite marked, and again the patient was barely able to recognize familiar objects.

Considerable ataxia was present in the finger to nose test, and this was more marked when he was told to place the ulnar fingers to the nose than when the other fingers were used. He complained continuously of a numb "breaking" feeling in his left hand, and also that he was very susceptible to temperature changes. When it was cold or warm his hand responded with extreme sensitiveness to changes in the weather from warm to cold or the reverse.

Subsequent examinations for sensation developed the interesting fact that touch sensation was altered more in the distal part than in the proximal parts of the fingers and that no difference in appreciation between the flexor and extensor surfaces of the hand existed. Touch sensation on the dorsum of the hand, however, was better appreciated as the thumb was approached. Sensation for pain was also more disturbed in the distal parts than in the proximal portions, but was distinctly better appreciated on the flexor surface of the hand than on the extensor surface. Different tests for temperature sensation did not seem to demonstrate any alterations from the normal. Applications of faradic electricity were much better appreciated on the palmar surface than on the extensor surface.

In one examination when the relaxation of the left hand was almost perfect it seemed as if the loss of the sense of position was more marked when the fingers were bent backward, that is, to the extensor side, than when they were bent forward to the palmar. This observation, however, was only made once. It may be said in passing that it is most difficult to obtain complete relaxation of the hand, and it is only when this is fully obtained, that the test for the sense of position is of positive value. It is our practice when testing for the sense of position to grasp the patient's hand at the wrist and ask him to completely relax, so that the slightest voluntary attempt of the patient to move his hand can be appreciated by the examiner by the transmission of motion to the palpating hand.

With regard to other examinations it need only be said that Dr. Spiller's report as to the sensory conditions was fully confirmed.

Case 3.—Apoplectic attack with left hemianesthesia, more marked in the upper extremity; later limitation of sensory losses to the left upper limb; still later touch and pain more disturbed in the distal than in the proximal portions of the fingers and hand; touch and pain also better recognized on the radial than towards the ulnar side of the hand; the sensations of pressure, localization, position and movement, and stereognostic conception better preserved in the thumb and forefinger than in the other fingers.

This patient was exhibited by Dr. C. D. Camp before the April meeting of the Philadelphia Neurological Society. She was a woman, 43 years of age, who had had four children and four miscarriages, and had an alcoholic but no specific history. Eight days before her admission to the nervous wards of the Philadelphia General Hospital, April 17, 1906, where she was under the care of Dr. J. H. Lloyd, she became excited, ran a short distance, sat down and immediately afterwards, trying to get up, fell, owing to weakness of the left side. She was not unconscious, but had a "peculiar" feeling in the entire left half of the body.

When examined by Dr. Camp on her admission the patient did not manifest any motor weakness in any portion of the body. She had no ocular nor facial palsies, and the limbs were moved equally and well in all directions. The tendon reflexes were, however, increased on the left side, and a distinct Babinski response was obtained on this side. The reflexes on the other side were normal. The patient had almost complete loss of sensation for touch and pain and temperature over the whole left half of the body, this not being limited, however, to the median line. The sensory changes even at this time were more marked in the upper limb than in the other parts. Examination of the visual fields showed no alterations. No symptoms of hysteria were present.

In a subsequent examination by Dr. Camp sensation was not as much disturbed as before. In the course of several weeks almost complete return of sensation in the left lower limb and over the chest, abdomen and face had taken place. The loss of sensation was now limited to the left upper limb. Dr. Camp also found considerable ataxia and complete astereognosis in this hand.

When examined by one of us (Dr. Weisenburg), about three weeks after the stroke, there was practically no involvement of the motor functions on the left side. The reflexes on this side were still somewhat exaggerated, and the Babinski response was prompt. Sensation in all forms was considerably disturbed in the left upper limb, and very little, if at all, in the other parts of the body. The senses of touch and of pain were more disturbed in the distal than in the proximal parts. A closer examination showed that the senses, both of touch and pain, were better recognized on the radial than on the ulnar side of the forearm. As in case 2, these two sensations were better recognized as the radial side of the hand was approached, both on the flexor and extensor surfaces.

The patient was unable to recognize clearly an object placed in any portion of the hand, but she was able to appreciate the smoothness or hardness or density or angularity of all objects with the thumb and forefinger, but could not do this with the other three fingers. Similarly numbers drawn on the palm seemed to be somewhat better, if at all, appreciated over the radial side of the hand than over the ulnar.

The senses of pressure, of localization, of position, of movement were all better recognized as the radial side of the hand was approached. This differentiation was as well marked as in case 2. Considerable ataxia was also present in the affected hand, and this seemed more marked when the ulnar fingers were approached to the nose than when the index finger was used in the test.

Case 4.—Initial Jacksonian attack chiefly causing flexion and extension of the forearm; sudden loss of ability to extend the flexed index and middle fingers; two weeks later a similar transient affection of the ring and little fingers; paroxysmal paresthesia showing itself as a peculiar sensation passing along the outer or ulnar border of the arm and forearm; disappearance of the attacks in the ring and middle fingers in about four months; improvement for several months, then renewal of paresthesia now affecting the ulnar border of the forearm; inability to freely abduct the little finger independently of the ring finger; tremor and moderate ataxia of the right upper extremity as tested by index and little finger to nose tests, also by writing and other methods; slight hypesthesia to touch over the ulnar border of hand as tested with cotton, this disappearing as tests continued; pressure sense retained everywhere; disappearance of sensory phenomena for two months followed by another paresthetic attack affecting the mid dorsum of the hand, the entire index finger, half of the middle finger, and distal phalanx of the ring finger; sudden transient loss of ability to use the middle finger; no objective sensory impairment, but persistence of former tremor and ataxia; no astereognosis.

A man over sixty years of age six years before coming under observation, first had an attack of jerking or twitching of the right arm of brief duration. The arm was adducted, and the forearm flexed, and while so held the movement affected the entire limb, although it chiefly showed itself in slight extension and flexion of the forearm.

Later he was treated for an abscess of the gall bladder, a condition which probably had no connection with his attack of local spasm.

For five years he remained in good health, until four weeks before the time when he first consulted one of us (Dr. Mills), when he suddenly lost the power, or apparently lost the power, to extend the flexed index and middle fingers, the other fingers being little if at all affected. Two weeks later he had a somewhat similar affection of the ring and little fingers. He could flex these, but for a few hours he could not extend them after they were flexed. This condition passed away during the night.

At this time and since at intervals he has suffered from a local paresthesia. He described this paresthesia as a peculiar sensation passing down the outer side of the upper arm to the ulnar border of the forearm. The patient had had no headache, vertigo, vomiting or other symptoms, being, except as above stated, in good health.

The patient was seen by Dr. Mills at intervals for four months, and then not for seven months. During the first three or four months the attacks of flexor spasm gradually disappeared. For the next six months his improvement continued, at least as regards the flexor spasm and paresthesia and the use of the fingers and hand. He became able to write, which he could not do before, so that he could attend to his own personal letters. It was, however, necessary for him to form his letters carefully, writing slowly.

After six months elapsed he again had a renewal of the paresthesia, which at this time chiefly affected the ulnar border of his right forearm. He had no return of the flexor spasm and inability to extend the fingers, but a peculiar condition of the ring and little fingers with regard to voluntary movement came on. He could not abduct the little finger of the right hand as he could that of the left, keeping the other three fingers in contact. He could, however, if the first fingers were held together by a rubber band or by the hand, abduct and adduct the little finger slowly, but not with as much freedom as the little finger of the other hand. When he did not hold the other fingers together and attempted to abduct the little finger, the ring finger invariably went with it as if the two fingers were glued together.

He had some tremor of the right upper extremity, especially of the hand; also a little of the left, but very slightly marked as compared with that of the right. Testing for ataxia of the upper extremity by the ordinary finger to nose method, some atactic tremor or ataxia of the right upper extremity, but none of the left, was found to be present. Testing by touching the nose with the little finger, while the index, middle and ring fingers were held together by a rubber band, the atactic tremor was about the same as when the test was made with the index finger. A similar test made on the left showed no tremor, or one so slight as to be scarcely noticeable.

No changes in sensation could be determined by coarse or comparatively coarse methods. The upper extremities were carefully tested by gently touching the parts with cotton. No loss of sensation could be detected by the most delicate methods in the left upper extremity, and none in the right except over a very limited area on the ulnar side of the right hand. A small area over the right hypothenar eminence was hypesthetic, or at least transiently hypesthetic, the diminution of sensation disappearing as repeated tests were made. These repeated tests showed that when he was first examined by delicate touches, the impairment went to the line of the metacarpophalangeal articulation of the little finger; after the tests were continued for a time he began to discriminate in this area as in other parts of the hand.

Testing him by Head's method for pressure sense it was retained for slight pressure in the tactile hypesthetic area. The pain and temperature senses were also retained, and no astereognosis of any part of the hand could be made out.

The knee-jerk on the right side was diminished; on the left it was distinctly plus. Achilles jerks were present and equal on both sides.

For about two months after the above phenomena were noted, the patient again improved in the use of his hand, gradually regaining power to separate his little from his ring finger in a nearly normal manner. The ataxia remained, but he improved in his ability to write. After the two months, however, he had another seizure which showed itself in a sensation of numbness or tingling, which starting near the middle line of the dorsum of the hand, passed down, enveloping the index finger, the middle finger from its tip to about the second phalangeal articulation, and the distal phalanx of the ring finger. The thumb and other parts of the fingers and the hand remained normal as to sensation. This paresthesia remained, and about two days later his middle finger suddenly became weak so that objects could not be held by it. No paresthesia had returned in the little finger and ring finger and ulnar border of the hand and forearm. The little finger could now be separated volitionally from the ring finger, although this, as other movements of the fingers, was awkwardly performed. He still had ataxia or atactic tremor in the upper extremity, especially showing itself in the hand and finger tests. He wrote his letters well, but with difficulty and rather painful slowness.

The patient called attention to an interesting fact which illustrated the difference between the control of the little finger of the right or affected limb and that of the left. He could insert the little finger of the left hand into the ear and manipulate it without the slightest difficulty, but he always had difficulty in inserting the finger of the right hand into the ear, although when it was once placed in this position he could move it around apparently with the same force and ease as the finger of the other hand.

Careful examination for objective impairment of sensation showed no loss, even to light touches, and no errors of location. His chief affection of the hand and fingers seemed to be, as it had always been, paresthetic and ataxic.

These four cases show a few points of great interest in connection with the subject of our paper. With regard to the positions of the lesions, as necropsy was obtained in only one case, of course nothing that is absolute can be said. In the one case with necropsy, the lesion destructive of the cortex and subcortex, was almost entirely in the postcentral and sub-parietal convolutions, only invading precentrally to a slight extent in one position. In the second case, first recorded by Dr. Spiller, the presumption is in favor of a lesion posterior to the central fissure, and this is true also of the third. In the fourth case, presuming it to be due to a cortical cerebral lesion, this is probably situated in the postcentral convolution, invading somewhat precentrally and posteriorly towards the sub-parietal region.

In the four cases the tendency was shown by the sensory disorders to select the ulnar or the radial side of the hand. In three of them the impairment of sensation, and the astereognosis were more persistent and decided in the middle, ring and small fingers and the ulnar portions of the hand. The thumb and index fingers were the first to improve and the last to succumb to persistent sensory disorder.

In all of the cases touch and pain senses were more disturbed distally than proximally in the fingers and hand.

In the first three of the four cases the sensations of touch and pain were more disturbed on the extensor or dorsal surface of the hand than on the flexor or palmar surface. This is an observation, which so far as we knew, at the time of the examination, had not been previously made.

In one of the cases (case 2) when the hand and forearm were totally relaxed, the patient had not the sensation of position and movement when the fingers were bent outward (extended), although he recognized position and movement when the fingers were bent inward (flexed). This is also, so far as we know, an observation not before made.

In the fourth case, which is one of great interest, the diagnosis of a cortical lesion, largely parietal, may of course be questioned, but a thorough consideration of the mode in which the sensory and paretic attacks in the upper extremity occurred, the persistence of ataxia in this extremity, although this was not marked, and the absence of positive symptoms which might relegate the case to the cerebellum, spinal cord or periphery would seem to make the diagnosis of a cerebral lesion or condition necessary. One of the most interesting points in this case was the absence of astereognosis, although ataxia was present and persistent, and paresthetic attacks recurred. The case lends some support to the view of the authors that the stereognostic zone is independent of the areas both of cutaneous and muscular sensibility, and also of the motor area.

It might be worth while in this connection to say a few words about the question of the existence of separate stereognostic and sensory areas from the standpoint of the development of function in the individual. Some recorded observations have shown that in children up to a certain age, while the sensations of touch, pain and temperature, are evidently recognized, topognosis, or the sense of location, stereognosis, and to some extent the musculo-arthroidal sense are wanting. These functions and their anatomical substrata develop as the infant becomes the child and the child the young adult. These facts lend support to the view taken by the writers that the localization of cutaneous sensibility is close to the motor zone, while the musculo-arthroidal sense and stereognostic conception are probably, as would be expected, in the concrete memory field or posterior association region or closely adjoining this region.

It is noteworthy in our four personal observations, in several of the nine observations collected by us, and also in the cases recorded by Russell and Horsley, to which attention will be presently called, that the impairment or loss of sensation in cases remaining under observation was invariably more persistent (unless the entire hand remained anesthetic), in the ulnar or postaxial portion of the limb. This is an observation of interest and one calling for explanation. It is probable that this lies at least in part in the fact that the cortical centres, sensory, stereognostic and motor, for those functions which are most frequently active in the individual have larger areas of representation, and that these are more deeply organized.

The earlier return of sensation or its greater persistence in flexor or palmar surface of the fingers and hand, and the more pronounced and persistent loss of the sense of position and movement in extension have a similar explanation.

The second proposition of this paper is that every muscle or group of muscles producing movement or movements which are represented by separate centres in the cortex is topographically related to a segment of the skin, which has also a definite cortical centre, this centre being correlated anatomically and functionally with the motor centre.

We believe that cases of limited sensory disorder due to lesions of the cerebral cortex or subcortex can be differentiated from the sensory affections of spinal, neural and hysterical origin, and also from anesthesias due to lesions of the thalamus and internal capsule, although this differentiation must, in some instances at least, be made by a study of concomitant clinical phenomena.

Some experiments in hypnotism may give a clue or a partial clue to the manner in which the cutaneous periphery is cortically represented and peripherally correlated to the musculo-arthroidal apparatus. To illustrate our meaning we might cite the following quotation from Heidenhain:

"Stimulation of different parts of the skin of the trunk constantly produces localized reflex movements; in the determination of these Dr. Born has kindly assisted me.

"Gently stroking the skin of the back at the sides of the spinal processes of the upper dorsal vertebræ causes elevation of the arms, with simultaneous slight flexion, so that the hands tend to meet above the head.

"Stimulation over lower dorsal vertebræ produced contraction of latissimus dorsi and rhomboidei, with resulting powerful backward movement of both arms and simultaneous flexion.

"Stimulation over the last dorsal and first lumbar vertebræ caused tonic contraction of the whole erector spinæ, with simultaneous elevation of the ribs (levatores costarum, intercostales) without accompanying contraction of the diaphragm, and hence passive sinking in of the abdominal muscles.

"Stimulation over the lower lumbar vertebræ and sacral region, the person being seated, caused contraction of flexors of leg, then of the ileo-psoas, and hence, the thigh being fixed owing to the sitting posture, the trunk was drawn down towards the thigh.

"Stimulation of the skin near the sternum: strong contraction of the pectoralis major, so that the arm was drawn to thorax; simultaneous stimulation of the extensors of arm.

"These reflex movements, which depend on the spinal cord and the medulla oblongata, represent a new series of phenomena in hypnotized persons, for previously only imitation movements, or such movements as had been passively induced by the operator, were known.

"It is certain that methodical investigation of the whole surface of the body would lead to the discovery of many more reflex relations."[14]

It will be seen in these experiments that the portions of the skin on which the stroking experiments were made were usually topographically correlated to the muscles or to some portions of the muscles through the instrumentality of which the movements resulting were performed.

We are not certain as to the exact state of the central nervous system during the existence of hypnosis. It is generally believed, however, that higher levels of the nervous system are in abeyance, that the activity of certain cortical cells is for the time being inhibited. The hypnotized subject is more or less lethargic and anesthetic because of the withdrawal through suggestion of the centres concerned with sensory and other forms of recognition. Persistent stimulation of certain areas of the skin unlooses the inhibited cerebral sensory centres and allows the cerebral reflex arc to be completed, the movements resulting being represented in segments of the spinal cord and re-represented in the cortex by cortical motor centres which are correlated to the sensory centres which have been called into action.

It is not probable that the areas of the skin which are separately represented in the cortex are in every instance exactly over the muscles, the cortical centres for whose movements are correlated to the cortical sensory areas, but skin and muscles, sometimes at least, bear this somewhat exact relation to each other; in other instances it may be that the cutaneous zone is related to certain portions of the muscles (as to their origins or insertions) concerned in the movements elicited. We do not know the exact topographical relations of correlated areas of the skin and muscular groups, but that they bear some relation like that above suggested, cannot be doubted.

To make our view as clear as possible we might cite some illustrations. The muscles performing certain very specialized movements, like that of flexion or of extension of the index finger, of adduction of the thumb, of ulnar extension of the hand at the wrist, or of flexion of the forearm at the elbow, have certain topographical relations with cutaneous areas. For the most part these cutaneous areas are over the muscles, or over the muscles, tendons and joints, concerned in the performance of the specialized movements. These cutaneous sensory areas are separately represented in the cortex, and the separate sensory areas of the cortex in turn are anatomically and functionally correlated with cortical motor centres.

The above views were presented by one of us (Dr. Mills) at the meeting of the American Neurological Association in St. Louis in September, 1904, and were prepared for publication in the present paper before the appearance of the paper by Russell and Horsley, to which attention will be presently called in detail. In a recent paper by one of us[15] our views with regard to the peripheral correlations between skin areas and muscular groups were presented as follows:

"In the periphery, segments of the skin are over or closely related topographically to the muscles producing movements which are represented in the motor cortex by centres which are connected with cortical sensory centres representing these cutaneous segments or areas. The segment of the skin covering the muscles producing adduction of the thumb, for instance, is represented in the cortex by a centre which is distinct but neuronally associated with the cortical centre for this movement of the thumb."

It will be seen that to a certain extent this view is in accord with the theory of Russell and Horsley of spinal segmental rerepresentation in the cortex. Preaxial, postaxial and midaxial cutaneous areas in the upper limb, for instance, may contain within them cutaneous subareas, each of which is represented by a cortical centre, and each of which is more or less correlated in position and extent to a preaxial, postaxial or midaxial muscle or muscular group. It is probable, however, that the areas of the skin represented in the cortex do not arrange themselves in the well-defined strips which correspond with the segmental or nerve root distributions. We may eventually get the diagnostic clues which will enable us to separate the spinal, neural and cortical areas of anesthesia by studying the differences in their respective topographical zones in patients with cortical, spinal or neural lesions.

The paper by Russell and Horsley[16] is on the apparent re-representation in the cerebral cortex of the type of sensory representation as it exists in the spinal cord.

The authors first refer to the differing views regarding separate or identical sensory and motor representation, adhering to the view that the areas of cortical representation of motion and sensation, or at least tactility, are identical or coincident. They also apparently adhere to the view, now largely disproved, that sensation and motion are represented almost equally on both sides of the central fissure. Horsley, as indicated in this contribution. has for many years believed that the type of sensory cortical representation is segmental.

It has been pointed out at the beginning of this paper that one of us has held for many years to the view that the representation of cutaneous and muscular sensibility in the cortex is subdivided after the manner of motor representation, the areas of representation, motor and sensory, being not anatomically identical. It was held for instance, in 1895, that differentiated areas and subareas for sensation were present in the cortex and that no part of the brain was more likely to contain these differentiated areas for sensation than the gyrus fornicatus, the hippocampal gyrus, the precuneus and the post-parietal convolutions.[17]

Neither of us sees any reason for changing the position taken, with the exception that we would now exclude from the region of cutaneous and muscular sensibility the hippocampal convolution and would include in it the postcentral convolution, and probably also the posterior and inferior portions of the paracentral lobule.

Russell and Horsley admit that the facts collected and the experimental investigations made which point to conjoint sensory and motor localization were largely, if not altogether, based upon an acceptation of the view that the motor cortex was post-Rolandic as well as pre-Rolandic. A view which they now admit is called into question by the researches of Campbell[18] and Brodman[19], although they fail to refer to the investigations of Grünbaum and

Sherrington[20] and the paper on cortical faradization of the human brain, published in this country by one of us and Dr. Frazier[21], and also to other American papers pointing to the sensory and stereognostic functions of the parietal cortex.

Discussing the question of separate motor and sensory localization, Russell and Horsley say "that the view has appeared to them to be contrary to the evolution of the nervous system, since from the point of view of function, every part of the central projection apparatus must be sensori-motor, and from the morphological standpoint, all the homologies of the structure of the pallium compared with that of the lower nerve centres establish the same principle."

This reasoning does not appeal to us, for while it is true that sensation and motion are always represented in a complete reflex arc, spinal or cerebral, it does not follow that each of these functions is represented equally or in the same manner in the same part of this arc. While it is also true that in the lower forms of animal life an afferent fibre, a nerve cell, and an efferent fibre represent the completed nervous system, in the differentiation of the nervous system, as evolution proceeds, such readjustments and intercalations take place that sensory and motor centres become separated in the spinal cord at basal levels of the brain and in the cortex. The most reasonable view, as well as that which we believe is supported by carefully sifted facts, is that the correlated sensory and motor areas are in distinct, although it may be in adjacent portions of the cerebral surface. This is in accordance with the usual method of anatomical and morphological development. As centres and areas are added in connection with the development of higher and higher functions, the additions are made by surface expansion rather than by superimposition of layers. The convoluted cerebral surface assumes its configuration in obedience to this law of surface expansion and elaboration to accommodate centres endowed with different functions.

Russell and Horsley (always taking for granted that the cortex is sensori-motor and on both sides of the central fissure) show that the sort of sensation disturbed in the experiments in cases to which they refer was (1) slight tactility; (2) topognosis; (3) general orientation of the paretic part; (4) occasional pain. By topognosis is meant false localization of the spot touched. They found that this disturbance of sensation was in the direction of proximality; in other words, the point indicated was nearer the body than that absolutely touched. The error was regarded as segmental. In a case of severe or extensive cortical lesion the patient, when touched lightly on a terminal phalanx, indicated a spot on the dorsum of the metacarpus, and in a less severe case on the next phalanx or the next but one.

We are distinctly in conflict with the view expressed in the paper under discussion that stereognosis is represented all over the motor cortex. We believe that this function has a separate cortical habitation. One of us, and others in this country and in Germany, have published facts which seem to us convincing as to the cortical area of stereognostic conception being posteroparietal in situation. Cases of cortical or cortico-subcortical paresis without astereognosis and cases of cerebral astereognosis without paresis are now on record.

We use the term stereognostic conception by preference, believing that this cerebral function or process in its typical form belongs in the field of concrete conceptual activity. The stereognostic region is a subarea of the concrete memory field.

The most interesting portion of the article by Russell and Horsley is that which relates to the apparent re-representation of the spinal cord metameric type of sensory topography in the cerebral pallium. The spinal segments or metameres, as is well known, are in direct relation with certain nerve roots, and these nerve roots supply the cutaneous periphery in a special manner which differs from the peripheral nerve distribution. Speaking generally, the nerve root distribution to the limbs, especially to the upper limbs, is in a series of longitudinal strips as shown in the well known diagrams of Edinger and others. Briefly stated, the view of Russell and Horsley is that these strips of skin represented by the nerve roots and spinal segments or metameres are re-represented in the cerebral cortex.

Many years since, one of us in an article on spinal localization called attention to the observations and work of Ross[23], Paterson[24], Herringham[25], Thorburn[26], and others in this field. Russell and Horsley refer especially to the observations of Ross and Paterson, and also to the later work of Grünbaum and Sherrington[27] and of Head.[28]

Ross introduced the terms preaxial and postaxial. The preaxial portion of a limb is the part situated on the internal or inferior aspect,—in the arm on the radial and in the leg on the tibial side; while the postaxial subdivision of the limb is situated posterior to the long axis,—in the arm on the ulnar and in the leg on the fibular side. The following quotation from the paper on spinal localization will serve to make the matter clear:

"Sensory segmental localization follows certain laws. If the upper extremity, according to Ross, be placed in the embryological position—that is, with the thumb directed outward and upward, and palm forward—the preaxial border from the tip of the shoulder down to the metacarpophalangeal articulations of the index finger and thumb is supplied by the fifth cervical root, and the postaxial border, from the axilla to the finger-tips inclusive, is supplied by the humeral branch of the second, the first dorsal, and the eighth cervical nerves. Observations made in cases of disease of the cauda equina have also convinced Dr. Ross tha. if the lower extremities be also placed in the embryological or tailor position, the preaxial border is supplied by the cutaneous nerves of the four upper lumbar nerves, and the postaxial by the coccygeal and sacral sensory nerves. He has concluded also that the most distal parts of the preaxial border were supplied from the lower of the four lumbar nerves, and of the postaxial border by the higher sacral nerves.

"The chief law of sensory distribution, as worked out by Herringham and adopted by Ross, is as follows:

"A. Of two spots on the skin, that which is nearer the preaxial border tends to be supplied by the higher nerve.

"B. Of two spots in the preaxial area, the lower tends to be supplied by the lower nerve, and of two spots in the postaxial area, the lower tends to be supplied by the higher nerve."

Russell and Horsley, also following Ross, define the terms preaxial and postaxial particularly as applied to the upper limbs, showing that the preaxial portion of the limb is supplied by the fifth, sixth and seventh cervical nerves, and the postaxial by the eighth cervical, and the first and second dorsal or thoracic nerves. They believe also that the central line or axis of the limb probably has a special morphological significance, and a special cortical representation.

As evidence of this they present two interesting cases; one unreported case of Dr. James Taylor and the other of Dr. T. Grainger Stewart[29], recorded in 1894.

In Taylor's case the aura of the Jacksonian attacks first began with sensations of pins and needles in the third and fourth fingers of the left hand. In this case a subcortical tumor was removed from beneath the postcentral convolution and forward to the island of Reil. Seventy-three days after operation, examination by medium pressure with pin point showed anesthesia of the dorsum of the hand and proximal errors towards the mid axis.

In Dr. Stewart's case sensation was disturbed in the entire upper extremity, except in a narrow strip in about the mid axis of the dorsal and ventral surfaces. Russell and Horsley hold that these cases and others of a similar character support the inference which naturally arises from the acceptance of preaxial and postaxial representation in the pallium, and that the mid axis of the limb is also there represented.

Russell and Horsley cite the notes of five cases bearing upon the question of preaxial and postaxial subdivisional representation in the cortex. We append the notes of these cases, largely in the language of the writers, simply somewhat condensing them and here and there changing the nomenclature to correspond to that used in this paper.

Their first case, one of Dr. Beevor's, was a man 42 years old, with pachymeningitis involving the right cortex over an area posterior to the central fissure and above the Sylvian fissure, the area including the lower half of the postcentral and the whole of the inferior parietal gyres. "The postero-inferior angle of the part removed included both sides of the posterior third of the Sylvian fissure. The depth of the incision was about 2 cm., *i.e.*, the floor of the lesion was composed of corona radiata.

A. Before operation. Slight diminution of sensibility to pain over hand and forearm to elbow, to camel-hair touch on fingers of the left hand. Loss of sense of position in fourth and fifth fingers, marked astereognosis. Postaxial error of localization on dorsum of middle, ring and little fingers of the left hand. On the palmar surface there was no error. On the dorsum of the left hand and wrist the topognostic error was proximal. The errors were always much greater after a fit, and gradually diminished during the succeeding ten to fourteen days.

B. After operation. (1) Immediately after operation, complete hemianesthesia of tactility and thermal sense, hemianalgesia of face, upper limb and trunk, relative on lower limb, hemianopsia, hemimotor paralysis of arm and face, slightly of leg.

(2) Fourteen days after operation, analgesia less marked. Visual fields restored; shoulder and elbow movements restored; also slight power of flexion and extension of the wrist and fingers. Complete loss of sense of position of the upper limb.

(3) Twenty-eight days after operation. Returning postaxial estimation of position of hand.

The second case of Russell and Horsley (case of Dr. Risien Russell) is recorded as follows: M. B., 40 years old. Glioma (mass removed 5 cm. longitudinal axis, 4 cm. frontal axis, 3 cm. vertical depth) situated immediately subcortical to the junction of the leg and arm centres in left hemisphere. The lesion left by the removal of the growth involved both the central ascending gyres; and extended into the corona radiata to a depth of from 3 to 5 cm.

A. Before operation. Soon after a Jacksonian fit, beginning in the foot and involving whole side and limbs, there was present in the right hand definite atopognosis (proximal error) astereognosis and hemiparesis of right side.

B. After operation. (1) Thirteen weeks after operation. Marked postaxial localization of all light touches on dorsum of digits, as well as topognostic proximal error.

(2) Sixteen weeks after operation. No analgesia, no loss of pain or temperature sense. Defective sense of light touches on dorsum of digits, normal on forearm, very marked proximal atopognostic error on digits and distal error on dorsum of carpus and matacarpus. The error was preaxial on the fourth and fifth digits; and postaxial on the first and second digits. When tested with Horsley's divided plate the postaxial error between the two index fingers was extremely marked.

The third case of the series was one of Sir Victor Horsley. J. C., male, age 56 years. Operation, October 21, 1905. A small myxoma about 3 cm. in diameter situated in the middle third of ascending parietal gyrus was found. There was considerable amount of softening in the corona radiata beneath the cortex, which certainly involved the fibres of the ascending frontal as well as the ascending parietal gyrus and probably also the fibres of the paracentral lobule.

A. Before operation. Hemiparesis of upper limb. Definite atopognosis proximal, in right hand.

B. Twenty-four days after operation. Anesthesia to light cotton wool touches all over dorsum of all digits, pain and temperature sense normal.

The topognostic error was preaxial on the dorsum of the digits as well as proximal. The latter error occurred as far as the carpus.

The fourth case was also one of Sir Victor Horsley's. G. P., male, aged 62 years. A small glioma occupying the breadth of the right ascending frontal gyrus and extending to the anterior wall of the fissure of Rolando. The corona radiata was compressed tion of the tumor involved the anterior third of the width of the slightly, but nowhere softened. The lesion made by the extirpa- ascending parietal gyrus opposite the crater in the ascending frontal gyrus.

A. Before operation. Marked atopognosis in the left hand (dorsum of digits).

B. After operation. Marked proximal atopognostic error of all digits of left hand. Very marked postaxial error of the same digits.

The fifth case of Russell and Horsley was a patient of Dr. Aldren Turner. W. D., male, 22 years old. At autopsy (a) Small tubercle ¾ cm. in diameter growing from pia at junction of posterior part of second frontal, with ascending frontal in left hemisphere. (b) Similar tubercle 1½ cm. diameter in mid third of post central convolution on left hemisphere, and another of about the same size at extreme upper limit of post central convolution. A fourth in right hemisphere ¾ cm. in diameter in first frontal convolution.

Case of multiple lesions of the kinesthetic area.

Attacks of spasmodic twitchings in the fourth and fifth fingers of right hand, and simultaneously of eyelids.

Slight motor paresis of the fourth and fifth digits.

Slight anesthesia to light touches on the fourth and fifth digits.

Error of topognosis very slightly distal and preaxial on the fourth and fifth digits.

In the first case of Russell and Horsley, the observations with regard to the radial or preaxial portion of the limb are similar to those recorded by us in our cases, and also by some of the observers cited by us. Russell and Horsley also note a fact to which we have directed attention in commenting on our own cases, that the errors of sensation were less marked on the palmar than on the dorsal surfaces. They also record the earlier preaxial or radial return of sensation and of stereognosis.

In the second case the distinction between postaxial and preaxial sensory disturbance is emphasized, and also between the dorsal and palmar recognition. In this case, as in the others, the differences between proximality and distality in the areas noted are given. In cases studied by us we have not made any observations on this interesting point. In the fourth of our original cases, an effort was made to determine whether there was any difference proximally and distally without any result, although as will be recalled, the impairment of sensation disappeared as the ulnar or preaxial border of the hand was receded from. In this case the impairment of tactile sensation was very slight. In two other cases some experiments were made as to proximal or distal errors, but without result. In the third case of Russell and Horsley preaxial and dorsal impairment was greater, and proximal errors prevailed as in the other cases.

In the fourth case the preponderance of sensory impairment was postaxial, dorsal and proximal.

In the fifth case the anesthesia was more marked postaxially, that is, in the fourth and fifth fingers, and in these fingers it was preaxial and distal.

Note.—While this paper is passing through the press, a case illustrating some of the points discussed in it, especially the existence of a separate cortical area for stereognosis, and the subdivision of this area, has come under our notice. This patient was under the care of Dr. R. S. Dorsett, of Philadelphia, with whom the patient was seen in consultation by Dr. Weisenburg, and subsequently by Dr. Mills after the admission of the patient to the hospital of the University of Pennsylvania.

The patient, a man thirty-two years old, with no history either of alcoholism or syphilis, was perfectly well until three weeks before coming under observation, when he awoke during the night with a pain in the left side of the back of the head and in the same side of the neck, this pain disappearing the following morning. Two days after this, he began to complain of a numb, dead feeling in his left arm, followed in a day by similar sensations over the left chest and abdomen and the left leg. These sensations have persisted. About two weeks after the onset of these sensations he noticed that the grip in his left hand was not as good as before when his attention and eyes were directed elsewhere than to the object grasped. He has never had headache, nausea, vomiting or any disturbance in his eyes.

When examined his eyes and cranial nerves were found to be in a normal condition. The grip of the left hand, when his attention was called away, was not as good as when he was looking directly at his hand, in which case it was normal. The left leg, like the face and arm, showed no weakness. The reflexes were somewhat prompt, especially on the left side, but no Babinski was present. Sensation for touch, pain and temperature, and bone sensation were normal over the left side and everywhere. The senses of position and movement were lost or greatly impaired in the fingers of the left hand, the loss becoming less as the thumb and forefinger were approached. To a less extent the sense of location was disturbed; more so, as the radial side of the hand was approached. The sense of pressure was normal. He could not recognize any object placed in or manipulated by his left hand, the astereognosis being absolute. The hardness or softness of an object, or its surface contour could however be recognized, but only by the tips of his forefinger and thumb, especially the latter.

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ABSCESS OF THE BRAIN.[1]

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The diagnosis of cerebral abscess depends chiefly upon the signs of some more or less rapidly developing lesion of the brain, with the discovery of a purulent process in the body, or of a wound of the head. Abscess of the brain without any evidence of pus elsewhere may be extremely difficult to diagnose. The cerebral signs are much the same as those of tumor of the brain. The choked disc may occur in either condition, but is more likely to be intense in tumor and to occur earlier, but I have observed choked disc of high grade in abscess.

The temperature and pulse are likely to be subnormal in abscess, but they may be normal or even above normal, especially if the abscess is complicated by meningitis. We should not depend too much on the condition of the pulse and temperature. The temperature is more likely to rise in the terminal stage of abscess, but a rise may occur at any period and be caused by the primary suppurative process, as a superficial wound or purulent pulmonary disease. The stupor, in my experience, is usually greater in advanced stages of abscess than in tumor, and yet an abscess may be latent or nearly latent a long time, and stupor may be pronounced with brain tumor. It is not uncommon to find slow cerebration preceding stupor in either condition. In many cases the diagnosis must be in doubt; but given a case of long-standing purulent otitis media, with recently developed signs of disease of the temporal lobe or cerebellum, the diagnosis of abscess becomes more positive. High leukocytosis speaks in favor of abscess or purulent meningitis in contrast with tumor. Where the cerebral symptoms appear after a purulent pulmonary process the diagnosis between tumor and abscess of the brain is difficult. Virchow is said to have been the first to call attention to the frequency of abscess following a purulent affection of the lungs. Still more difficult is the determination as to whether purulent meningitis or brain abscess is present, and often the two conditions are associated in the same person. The diagnosis is important, because less can be hoped for from operation if purulent meningitis develops, whereas operation is demanded if the condition is one of abscess. Lumbar puncture[1] is of great value, as pus in the cerebrospinal fluid obtained by this means points to purulent meningitis; but purulent meningitis may exist although pus in the spinal fluid is not obtained. The presence of mononuclear or polynuclear cells in the cerebrospinal fluid is also of diagnostic value. Kernig's sign is of value as indicating meningitis, but is not pathognomonic, and may occur with abscess. Rigidity of the neck is more likely to occur in meningitis, but may occur with abscess. Where abscess is associated with purulent meningitis the prognosis is extremely grave.

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[1] Read by invitation before the Allegheny County Medical Society at Pittsburg, May 15, 1906.

The following case shows how difficult it may be to decide between cerebral abscess and cerebral hemorrhage:

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[1] An excellent presentation of lumbar puncture has recently been made by Kopetzky, Amer. Jour. of the Med. Sciences, April, 1906, p. 648.

A man, about forty years of age, was admitted to my service at the University Hospital, in an unconscious condition, March 20, 1906. No history was obtainable, except that he was known to be a chronic alcoholic, and had been unconscious on several occasions. He had been found in a stable, and brought from there to the hospital. No signs indicating an injury could be detected.

I examined him March 21. He had conjugate deviation of head and eyes to the left, and paralysis of the right upper and lower limbs. The temperature was above normal, 101°, and on one occasion 103°, until March 26, and remained normal until April 27, when it rose to 101°; it fell again to normal on April 30. I supposed the man had probably had a hemorrhage of the brain. No discharge from the ear was present. He improved somewhat, but never spoke. A copious purulent discharge from the left ear began April 28. His condition had improved by this time to such a degree that a more thorough examination could be made. He was still completely paralyzed in his right limbs and right side of his face; he had diminished sensation of pain on the right side, and had also right hemianopsia. He was evidently word deaf, and could not speak. He had a leukocytosis of 16,640. Lumbar puncture allowed 30 c.c. of perfectly clear fluid to be withdrawn, but a smear preparation of the sediment showed no cells. Kernig's sign and rigidity of the neck were not present.

Dr. Randall examined the ear and expressed the opinion that the discharge was caused by chronic suppurative otitis media and that it did not contain cerebrospinal fluid. There seemed to be no mastoid involvement.

Dr. Carpenter, in Dr. de Schweinitz's clinic, examined the eyes and reported that the man had bilateral neuroretinitis with hemorrhages. The swelling of the disc was not marked and the fundus disturbance was rather widespread. The veins were very much swollen.

On account of the purulent otitis media, the leukocytosis, the neuroretinitis, the symptoms pointing to a lesion in the left first temporal convolution and extending to the internal capsule and optic radiations (right hemiplegia, hemihypalgesia, hemianopsia, word deafness), I feared the patient might have a cerebral abscess, and requested Dr. Frazier to operate over the left first temporal convolution. A subdural hemorrhage was found in this region. The otitis media and the hemorrhage on the brain were probably the result of trauma. Had there been no otitis media the opening of the skull probably would not have been made, as the symptoms otherwise seemed to point to an inoperable intracerebral hemorrhage. The patient died two weeks after the operation, and a large hemorrhage was found within the left basal ganglia.

Headache and vomiting are common and early signs of abscess or tumor of the brain. Chill is a common symptom of abscess, and likewise is general muscular wasting, if the patient lives sufficiently long. Frequent yawning is not uncommon with brain abscess or tumor. I have seen it a number of times, and occurring as it does, when the patient is in marked stupor, it is a striking sign.

As an example of an unusual method of infection of the brain, I mention a case described to me by Dr. B. A. Randall. A boy was tossing up an umbrella, when one of the ribs penetrated the ethmoid bone through the nostril and caused cerebral abscess.

Abscess of the brain or purulent meningitis may readily occur in association with a purulent thrombus of the lateral sinus secondary to otitis media. Indeed, these conditions are sometimes produced in this way, although sinus thrombosis is not the most common cause of abscess. More frequently the infecting organism is carried directly into the brain substance. The diagnosis as to whether or not abscess complicates sinus thrombosis is of great importance, because it is desirable to leave the dura unopened if it can be done with safety. I have seen a case under the care of Dr. B. A.

Randall (Case I) in which this question arose. The notes are given in detail further on.

The right external jugular vein and some of its tributaries were ligated two inches below the angle of the jaw, by Dr. Randall, on account of thrombosis and the part above removed. The right lateral sinus was thrombosed. It was opened, the thrombus removed, and the sinus packed. The right ear had discharged purulent fluid for four years. Dr. Randall invited me to see the patient after this operation. The boy was in marked stupor, but could be aroused to answer questions. The pulse was about 65 and the temperature was normal. The patellar reflexes were absent. He had had some ataxia of his left limbs, but had not had any weakness of his limbs nor the Babinski sign. The symptoms were suggestive of cerebellar abscess, and the question therefore became very pressing as to whether the dura should be opened or not. The symptoms after the operation on the lateral sinus and jugular vein were very grave, but in the opinion of Dr. Randall and myself might be explained by the lesions already found, and to open the dura through an infected region would expose the boy to grave danger of intradural infection. The latter might be avoided by sterilizing the dura at the point of operation, and later by packing with iodoform gauze; but the penetrating powers of micro-organisms is great, and we were eager to avoid the risk of adding any complication in the serious condition of the little patient. Delay was therefore decided upon. The symptoms were carefully watched with the idea that any increase in them would justify the opening of the dura and searching for an intradural abscess. Just here I may remark that judicious delaying is one of the most difficult problems of cerebral surgery. Our course was justified, the patient slowly improved, and a few weeks ago I had the pleasure of seeing him in very good condition.

E. W. Roughton,[1] in reporting a case of cerebellar abscess, recently says: "Let me now tell you that much more severe and definite symptoms of intracranial disease may occur in cases where, so far as one can judge, there is extensive disease of the ear and mastoid process without any actual disease inside the skull; perhaps one is not justified in stating that there is no intracranial disease in these cases, but it is certain that they may recover after operative treatment limited entirely to the ear and mastoid process." He refers to the case of Sainsbury and Battle, in which the symptoms were severe, and constant headache, drowsiness, frequent vomiting, giddiness, inequality of pupils, diplopia, well-marked double optic neuritis, and pyrexia of an irregular type were present. Mastoidectomy was performed and the patient recovered completely. The cranial cavity was not opened. These writers believe that in all cases of ear disease with head symptoms sufficiently grave to call for operative interference and including even a severe optic neuritis, the patient should not be subjected to the major operation of craniotomy until the minor operation of clearing away all extracranial disease has been performed. I would urge, however, that the delay should not be long, as the symptoms mentioned above are very important.

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[1] Lancet, 1905, vol. ii, p. 1597.

It is astonishing that purulent otitis media may exist a long time and not cause brain abscess until after many years. Oppenheim speaks of a case in which the otitis media existed thirty-four years, and of another in which it existed forty-five years before abscess developed, and yet acute otitis media may cause abscess. According to Grunert, in 91 per cent. the process is chronic, in 9 per cent. acute. Schmidt and Gruber have reported cases in which the ear disease seemed to be cured before a brain abscess developed, and on the other hand Baginsky and Gluck have observed a case in which signs of brain abscess appeared on the twenty-second day after the commencement of the ear disease (cited by Oppenheim). I have had the opportunity of observing with Dr. Randall a case in which a temporal lobe abscess developed in a man sixty-eight years old (Case II), who had had middle ear disease most of his life. As the symptoms of brain abscess began only a few weeks before his death, it does not seem probable that he had had a latent abscess for a long period of time. Before the operation on the brain the pulse was 102 and the temperature 99° and a fraction; but this man did not have meningitis and had two abscesses near together in the temporal lobe, probably parts of one lesion, only one, however, was found at the operation and evacuated. The elevation of the pulse and temperature when I saw him was late in the disease, and this is sometimes the case in abscess.

Oppenheim, probably the first, has called attention to the occurrence of hemorrhagic encephalitis as the result of otitis media. The symptoms are likely to be those caused by lesions of the motor cortex.

Multiple abscesses make operation very uncertain, as there is no means of determining from the symptoms whether the patient has one or several abscesses, possibly communicating, and abscesses often are multiple. Abscess of the brain not infrequently is multilocular, and one or more pockets may escape detection, as in the case referred to above (Case II). Another difficulty the surgeon has to contend with is unusual density of the pus. In one of the cases I had the opportunity of studying with Dr. Randall the diagnosis of cerebellar abscesses seemed sufficiently clear to justify operation. This was done by Dr. Randall. The patient's temperature before operation was about normal; her pulse ranged from 64 to 108. Unfortunately the notes have been mislaid. She had lateral nystagmus in looking toward the side of the cerebellar abscess, and this sign I believed might be of value in determining the affected lobe, but I have not been able to find it in any other case, although I believe it has been observed a few times by others. The necropsy showed that Dr. Randall must have entered the abscess with the trocar, and yet no pus flowed. The lesson to be learned from this case is that the brain or cerebellum should be incised and not merely punctured at the place suspected of concealing an abscess, as little damage is done to a lateral lobe of the cerebellum by incision if the wound does not become infected; indeed, the cerebellum seems to be able to stand much rough handling, even the excision of a large part of a lateral lobe, without causing serious symptoms, as we have seen in a number of cases at the University Hospital.

Most of the cases of brain abscess I have collected for this paper have resulted from middle ear disease, but a case reported by Dr. C. K. Mills and myself, in 1896, was without discoverable primary lesion at the necropsy. The primary focus may produce no clinical signs.

C. H., a baker, forty-eight years of age, was admitted to the hospital January 30, 1896. He had previously been in fair health. His mother had never heard him complain of earache. During the summer of 1895 he suffered constantly from severe headache.

On December 20, 1895, he became unconscious and had three general convulsions. He remained unconscious two days, and when he regained consciousness it was noticed that he was partially paralyzed on the right side, and that he could not talk properly.

January 29, 1896, he had another attack of unconsciousness without convulsions, but with aphasia and paralysis. He was brought to the hospital next day. On admission he was in a condition of stupor; he did not speak when addressed and had almost total right-sided paralysis, incontinence of urine and feces, and entire loss of pain and touch sensations over the paralyzed side. By testing him with food brought toward his mouth from the right or left side it was noticed that he opened his mouth to receive the food only when it was brought from the left side. Likewise, when a pointed instrument was brought near either eye from the side, as if to touch it, he winked only when the motion was made in front of the left half of each eyeball. From these tests it was evident that right homonymous hemianopsia was present. He was found to have double papillitis, more marked in the left eye. His general condition gradually became worse. He occasionally complained of headache, which he did not localize.

On February 25, 1896, breathing was stertorous and the face dark red in color. The coma gradually increased until he died, twenty hours after the beginning of this attack. There was no evidence at any time of middle ear disease. During the entire time he was in the hospital, until just before death, respiration, pulse, and temperature presented no indications of the purulent process within the brain.

A horizontal section made just above the corpus callosum revealed an abscess on the left side in the centrum semiovale, and corresponding anteroposteriorly with the position of the optic thalamus, internal capsule, and part of the lenticular body. Both tympanic membranes were normal. No evidences of suppuration or perforation were present. The pleura, pericardium, heart, spleen, and kidneys were normal.

Microscopic examination of the pus from the cerebral abscess showed only the ordinary staphylococcus pyogenes aureus. There were about two ounces of pus in the abscess cavity and left ventricle. Both eyes were removed and examined by Dr. Oliver, and the posterior halves of the eyeballs showed well-marked choked discs, more decided on the left side; the swelling and inflammation of the nerve head and surrounding retina were extensive and covered at places with minute capillary hemorrhage.

The right hemiplegia and right homonymous hemianopsia made a diagnosis of some lesion at the posterior part of the left internal capsule probable. The convulsions may have been caused by rupture of the abscess into the left lateral ventricle. The patient seemed to be word deaf.

As cerebral abscess may occur after suppurative processes in the lungs, these organs were examined, but nothing was found, with the exception of spots of catarrhal pneumonia in both lungs. Suppuration was present only in the cerebrum.[1]

One of the cases coming under my observation, the clinical history and pathological material of which I have obtained from Dr. Chas. S. Potts, under whose care the patient was, shows that a cerebellar abscess may readily escape detection (Case III). The temperature varied very little from the normal, but the pulse was a little high. The patient was a man, aged fifty-seven years, somewhat dull mentally, and had been unable to walk for a year on account of vertigo, and had complained of headache about four months, and had ataxia in walking. He was in semi-coma two days before his death. Ear disease was not suspected, and there was little reason for a diagnosis of brain abscess; and yet a large and small abscess near together were found in the left cerebellar lobe. The posterior inferior cerebellar artery passing to this lobe was dilated and thrombosed, and it was evident that in this way the abscess had originated. While the abscess in this case was not entirely latent, the symptoms were not sufficient to justify a diagnosis during the life of the patient.

Cerebral abscess is usually regarded as more common than cerebellar, and as Henri Bourgeois[2] says, this may be because the latter is less often diagnosed, and less often operated upon; but I doubt the correctness of this statement. He refers to the statistics of Acland and Ballance, taken from the records of St. Thomas' Hospital from 1876 to 1894, and according to these, nine abscesses were in the temporal lobe and seventeen in the cerebellum, and two of the latter were associated with temporal lobe abscess. The same investigators have found that at the Hospital for Sick Children in thirty-five years two abscesses of the temporal lobe and four of the cerebellum occurred.

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[1] Mills and Spiller, Journal of Nervous and Mental Disease, September, 1896.

[2] Contribution a l'étude des abcès otitiques du cervelet. G. Steinheil, Paris, 1902.

Oppenheim[1] says that Pitt in 9000 necropsies found brain abscess in 56, of these 18 arose in the ear or temporal bone. Freitel in 6000 necropsies found abscess twenty-one times, and of these seven arose in the ear. Others regard one-half or more of the cases of brain abscess as arising in the ear. Gowers puts it at 42.5 per cent. Men have abscess two or three times as often as women. Le Fort and Lehmann's statistics of 458 cases of brain abscess are: Cerebrum, 327; cerebellum, 113; cerebrum and cerebellum, 11; pons, 5; cerebral peduncles, 1; fourth ventricle, 1. So that the relative frequency of cerebellar abscess to temporal lobe abscess is as one to three. Dr. Randall informs me that in his experience cerebellar abscess has been more frequent than cerebral.

Case IV, recently under my observation, shows how exceedingly difficult it may be to diagnose between abscess of the brain and purulent meningitis. A young man was admitted to my wards at the Philadelphia General Hospital, in marked stupor and paralyzed on the left side of the body. The temperature varied from 98.6° to 102° and was never subnormal; the pulse varied from 80 to 110; the respiration from 20 to 25, or occasionally 30. Cerebrospinal fluid obtained by lumbar puncture showed lymphocytes 4 per cent., but no organisms. Within a few days he was found to have a purulent discharge from his right ear. The diagnosis seemed to be abscess of the right temporal lobe, implicating the right internal capsule and thereby causing left hemiplegia, possibly associated with purulent meningitis, and secondary to otitis media. Dr. E. Laplace, whom I called in consultation, and I decided that the patient was unable to endure an operation. A day or two before his death rales were heard in the lungs. He had no abscess of the brain, but the upper part of the right cerebral hemisphere was covered with pus, as was also to a less extent the corresponding part of the left hemisphere; pus was also found at the base of the brain and in the lateral ventricles. Cultures taken at the necropsy revealed tubercle bacilli, and miliary tuberculosis of the lungs was found.

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[1] Der Hirnabscess, Nothnagel's System, Band ix, Theil 11.

CASE V, studied by Drs. Hoyt, Randall, Frazier, and myself, shows how difficult the diagnosis of cerebral abscess may be. The slight elevation of temperature suggested purulent meningitis; the slightly subnormal pulse, abscess; both, however, might indicate abscess. The presence of pus in each ear and in the nasal sinuses without localizing cerebral symptoms made the diagnosis of location difficult, but the marked stupor left no doubt in our minds that some intracranial lesion was present, and we believed that it was purulent chiefly because of the purulent processes in the bones of the skull. Optic neuritis, so valuable a sign of tumor or abscess, was not present. The pain was confined to the left temporal region, but this was an unreliable sign, for I have known localized pain of the head to be very misleading. However, because of its presence on the left side, and the more serious middle ear disease on the left side, the operation was performed on the left side. We fully recognized that the abscess might be in the right temporal lobe. The absence of word-deafness in this case did not alter our diagnosis, as abscess in the temporal lobe is usually in the lower part of the lobe and below the centre for word-hearing. In a left-handed man, as the patient was, it would not be likely to cause deafness even if it extended into the first left temporal convolution.

Dr. Hoyt, who first had charge of the case, and performed the necropsy, believed from the findings at the necropsy that the purulent meningitis was of recent origin, and the question arises, Could it have been caused by the free irrigation of the abscess cavity and the washing thereby of some of the infectious material into the meninges. [Much flocculent meningeal fluid was found before the abscess was irrigated.] An abscess cavity possibly should be irrigated, but there is danger thereby of producing a purulent meningitis. Aspiration would be a dangerous proceeding, as often the pus would not be thoroughly evacuated and in some cases would not flow freely through the needle. It seems well to avoid very intense irrigation of an abscess cavity, and it is possible that drainage with moderate irrigation would be sufficient, especially if after the operation the patient is made to lie with the opening downward, so that the pus may find free escape. Dr. Müller has suggested to me that sponging out the abscess as is done in the abdominal cavity might be sufficient.

Unusual and interesting cases of brain abscess are the following: A case of brain abscess with cure was reported by Randall and Potts[1] in 1903. After removing pus from the mastoid cavity a perforation of the dura, 2 mm. in width, was discovered, out of which pus was escaping. An incision through the dura exposed the brain substance, which showed no appearance of intracerebral abscess, but a few days later such an abscess was found and evacuated. A few days still later another abscess cavity was evacuated. The child recovered. There seems to have been a limited subdural abscess in this case associated with multiple intracerebral abscesses, and yet recovery occurred.

Pial abscess is uncommon, but Randall[2] reported a case in 1894 in which in association with extensive meningitis a small abscess cavity was found lying between the medulla oblongata, pons, and cerebellum; a smaller abscess occupied the corresponding position on the right, while a third, much larger, was found above the medulla oblongata. These three abscesses were within the pia. In this case the pulse was slow when the temperature was high. This case resembles an unpublished case seen by Dr. T. H. Weisenburg whose specimens I have seen.

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[1] Archives of Otology, 1903, No. 3, vol. xxxii.

[2] The University Medical Magazine, June, 1894.

The brain abscess is likely to be on the side of the middle ear disease and near the petrous portion of the temporal bone, but in some cases the discharging ear may not be the primary focus, and the other ear may have been the longer and more seriously affected, although at the time of observation there may be no discharge from this ear. In a case (Case III) reported by Stoll[1] the abscess was secondary to an acute otitis media, and was situated in the parietal lobe. The author gives as an explanation for this unusual occurrence that the infection must have been embolic in character, and the embolus so small that it was not arrested until it had wandered some distance from its origin.

Abscess of the brain developing in the course of typhoid fever has been observed by A. C. Brown,[2] and his seems to have been the first case of cerebral abscess caused by this disease. The lesion was supposed to be a tumor, and operation resulted in evacuation of the pus and recovery. McClintock[3] has reported brain abscess caused by the typhoid bacillus.

The cause of death in operations on cerebellar abscess, according to Barr and Nicoll,[4] in many cases is thrombosis of the veins of Galen, with distention of the ventricles and œdema of the brain. The fatal result may rapidly follow the operation or may be delayed for weeks or months, even when recovery from the effects of the abscess is supposed to have occurred.

Abscess following trauma of the head without injury of the skin and bone of the head may occur. Ehrnrooth[5] has reported such a case. Intracranial hemorrhage certainly may occur from trauma without fracture of the skull, and probably a damaged portion of the brain may offer a favorable soil for the growth of bacteria, but nevertheless bacteria must penetrate in some way.

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[1] American Journal of the Medical Sciences, February, 1906, p. 223.

[2] Edinburgh Med. Journal, new series, 1900, vol. viii, p. 228.

[3] American Journal of the Medical Sciences, April, 1902, p. 595.

[4] British Med. Journal, February 16, 1901, p. 390.

[5] Deutsche Zeit. f. Nerv., vol. xx, Nos. 1 and 2, p. 103.

Cassirer[1] has found reported in the literature sixteen cases of abscess of the pons and medulla oblongata, but of these only ten seem to him trustworthy. Two, or possibly three, were cases of abscess of the medulla oblongata; in the others both pons and oblongata, or pons alone, were implicated. He himself has reported a case. The reasons for the infrequency of abscess in this part of the brain, according to Cassirer, are the relative smallness of these parts, the less liability to injury, and the remoteness from the various cavities of the skull from which the primary infection comes. If a septic embolus is carried to the brain it is more likely to lodge in the left Sylvian artery. Possibly also the pons and medulla oblongata are more resistant to bacteria, as some have thought.

Abscess in each temporal lobe has been observed by Whitehead.[2] One abscess in the left temporal lobe was evacuated and eighteen months later the patient was well, but later cerebral symptoms developed. An operation revealed an abscess in the right temporal lobe, and this abscess was drained and recovery from the second operation also occurred. The patient had had a purulent discharge from each ear.

An interesting paper on cerebral abscess, which I read after my paper was completed, has recently been published by M. Allen Starr.[3]

RECORDS OF CASES.

In publishing the abbreviated records of the following cases I wish to thank my colleagues for permission to make use of their notes in connection with my own, especially Dr. B. A. Randall, with whom many of the cases were seen.

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[1] Archiv. f. Psych., vol. xxxvi, No. 1, p. 153.

[2] Lancet, February 13, 1904, p. 428.

[3] Medical Record, March 10, 1906.

CASE I.—*Sinus Thrombosis, with Symptoms of Brain Abscess.* (Service of Dr. Randall.) Philip W., aged fifteen years. *Chief Complaint.* Purulent discharge from middle ear followed by dizziness, headache, drowsiness and stupor with pain, induration, tenderness, and œdema over an area extending two and a half inches below the mastoid tip.

Four years ago he was struck over the right ear. Every winter since then he has had some discharge from his middle ear of a purulent nature. Two weeks ago this came on again as usual. Five days before admission he became suddenly dizzy and was obliged to take to his bed. He complains somewhat of pain below and behind the right ear, and has been in a semi-stuporous condition since, although able to answer questions intelligently.

Condition on Admission. Pupils are dilated and equal. Eye-grounds negative. Foul, light yellowish discharge from the external auditory meatus. No swelling, pain, or tenderness over the mastoid. Below and behind the mastoid extending down the neck there is an area (about two inches in diameter) which is tender, indurated, and œdematous. No pain in mastoid on percussion. Left hand and arm are slightly ataxic. Knee-jerks absent. Power in the arms and legs seems normal. No Babinski; no ankle clonus; leukocytes, 28,320.

Operation, December 4th. Under ether anesthesia, Dr. Randall at 12 M. made an incision over the right external juglar vein; it was found to be thrombosed down to a point two inches below the angle of the jaw where it was ligated, as were also some tributaries. The wound was closed with gauze drainage. An incision was then made over the mastoid down to the lateral sinus. The bone was much sclerosed, but bled freely. The sinus was found large, thick, and thrombosed. It was opened, the thrombus removed, and the sinus packed after considerable difficulty; no pus was found.

The middle ear was entered through the antrum and through-and-through drainage established. After thorough packing the wound was closed.

December 12th. Since operation he has been somewhat stuporous and apparently sleeping most of the day. His pulse remains steadily about 65. Temperature normal. Reflexes persistently absent. Upper portion of wound in neck is discharging a thin yellow fluid. Some induration in neck. He is nervous and irritable and his appetite is capricious.

Blood Count. Hæmoglobin, 55 per cent.; red blood corpuscles, 3,290,000; white blood corpuscles, 14,720.

December 18th. He has been steadily improving, does not sleep nearly so much; has been kept in sun parlor; is now sitting up; temperature normal, pulse rising, although still full. Appetite is good, sleeps well; knee-jerks, however, are persistently absent.

December 23d. Is now sitting up in a chair; wound does not pain. He is dressed every other day; seems much brighter and is gaining weight. Full recovery ensued.

Case II.—*Otitis Media of Long Standing, Symptoms of Brain Abscess of Short Duration.* April 17, 1901. Notes made by me in consultation on a case under the care of Dr. Randall. C. A., age sixty-eight years.

About January, 1901, the right ear was operated on by Dr. Randall and much pus and cholesteatoma masses were removed. Following this operation, and possibly before it, his mental condition was abnormal; he was, as expressed by one who had long known him, childish, but not irritable. His family have noticed for some weeks that the movements of the left face were somewhat impaired. About ten days ago the first signs of a distinct gradually developing paralysis was observed without apoplectic attack, in the left side of face and left upper and lower extremities. This paralysis never became complete. He dragged the left lower limb in walking. Yesterday he was in stupor for the first time, but was aroused when Dr. Randall visited him and he talked rationally for some time; he then went into stupor again and has remained in this condition since. He only once complained of feeling somewhat chilly. During the past week he has had considerable frontal and occipital headache, but no vomiting and no distinct vertigo or ataxia. I saw him this afternoon (April 17th) and found him in profound stupor, so that he could not be aroused. The right limbs were moved voluntarily, but the left were only imperfectly moved. Involuntary jerking movements were seen in the left toes and left fingers, suggesting, in connection with other symptoms, irritation of the central motor tract. The left upper and lower limbs were very spastic. The reflexes in the upper and lower limbs were increased on both sides, but were checked somewhat by the spasticity of the left limbs. The left knee-jerk was more exaggerated than the right. The Babinski reflex was present on each side, but more marked on the left side. The left pupil reacted promptly to light. Hemianopsia could not be tested for on account of the stupor. The limbs were withdrawn when they were stuck with a pin, but the left upper and lower limbs were not so freely moved. Yawning was very frequent. No rigidity of head was noticed. Pulse 102, just before operation was 120; temperature, 99°. Neither side of the face was drawn. He never had slowness of pulse.

Dr. Randall adds to my notes that the patient had come under his care January 9, 1901, with chronic suppuration of the right ear, attic caries and cholesteatoma, having been long under Dr. Risley's care. Exenteration of tympanic cavities at the University Hospital, January 12, 1901; discharged, February 2, 1901, from hospital with wound healed and cavities dermatizing. Progress soon halted. Some weakness of left arm and mental slowness developed about March 10, 1901, previous to which he went about freely. These symptoms increased and on March 16th he became unconscious. A cerebral abscess in the sphenotemporal lobe was evacuated the evening of March 17th. He did not recover consciousness; the wound was dressed on the 19th and finger sought further abscess in vain. He died March 20, 1901.

Autopsy showed second abscess adjacent.

Case III.—*Cerebellar Abscess from Occlusion of Posterior Inferior Cerebellar Artery, Unrecognized during Patient's Life.* H. R. was admitted to the service of Dr. C. S. Potts, in the Philadelphia General Hospital, July 18, 1904, and died August 22, 1904, aged fifty-seven years. Dr. Potts' notes, August 20, 1904, are as follows:

Family History. Patient states that six months ago his hair began to fall out. Two months after this he began to have headaches, which persisted throughout the day. He also noticed about this time some difficulty in walking; he would stumble occasionally. At times he would have pains throughout the body.

He seems somewhat dull, but answers questions. He has been unable to walk for the past year on account of attacks of giddiness. He complains of headache. The left pupil reacts to light and in accommodation. No paralysis of the cranial nerves is found. The upper and lower limbs are normal in strength. He states that except for attacks of giddiness he can walk well. Knee-jerks and tendo Achillis jerks are present and normal. The plantar reflex is present, but not active, though all the toes flex. Sensations to touch and pain are normal. Stereognosis is preserved. He touches the tip of the nose with the index finger of either side very awkwardly.

Duration of illness two days.

Symptoms. Semi-comatose state; headache, backache, inco-ordination; throat and skin dry; polyuria; constipation; epigastric pain; coldness of hands and feet; always thirsty.

Autopsy. August 23, 1904. Embolism and aneurysm of left posterior inferior cerebellar artery; fibrosis of left cerebellar hemisphere, two abscesses of the left cerebellar lobe.

Case IV.—*Tuberculous Meningitis Causing Symptoms of Brain Abscess.* Christian Grandin, aged twenty-six years; white; was admitted to the Philadelphia General Hospital, December 27, 1905, and came into my service January 1, 1906. Died January 17, 1906.

Chief Complaint. Left hemiplegia.

Present Illness. About five weeks ago he complained of weakness in the left arm and leg. This weakness was irregular in its duration and would come and go. Sensation was also lost at times. Finally, about a week later, he was compelled to go to bed, but complete loss of power did not occur until one week ago, at the same time the mind became affected. The brother says that the patient suffered from headaches during the past week.

General Appearance. The patient is a well-developed adult male, very apathetic and in a semi-stuporous state. He cannot answer questions intelligently but does respond when spoken to. His response consists usually of grunts, but occasionally he gives an incoherent answer. While being spoken to he uses the right arm continuously, takes the comb out of the interpreter's jacket and puts it to his head and tries to comb his hair and then returns it to the owner. When given a hand mirror with its back toward him, he turns it around and looks at himself and returns it to the owner.

Pupils are equal, right slightly larger; react to light; impossible to test for accommodation and convergence, also for associated movements. Ptosis of both eyelids is present, and is more marked on the right side. When he sleeps the left eyelids are not completely closed. When he opens his mouth and shows his teeth the mouth is drawn toward the right side, and the nasolabial fold on the right side is much more prominent than that on the left side. The right corner of the mouth is also drawn up. There is no apparent weakness of the occipitofrontalis and corrugator muscles, although when he was asked to look upward this morning there was an apparent weakness of the muscles on the left side. The tongue is moist and coated and shows slight tremor and deviates toward the left. Pulse varies in rate, at times fairly slow and then increases in rate, is full, comes up quickly and recedes quickly; tension good.

The right arm is well-developed; the power and motion are good. The left arm hangs lifeless at his side, but is not atrophied. The right leg is fairly well-developed and power is apparently good. The left leg is fairly well-developed, but power is lost; no atrophy or spasticity present. The tendon reflexes are prompt. The Babinski reflex is obtained on both sides.

He is semi-delirious; talks incoherently; he has an old perforation in attic of ear drum; no apparent evidence of neurosis; no bulging of upper back wall; chronic nasopharyngitis; no evidence of sinus trouble obtainable; ear discharging pus. Pulse rapid, full, of high tension. Vessels of conjunctiva of right eye are injected. Temperature varied from 98.6° to 102°, and was never subnormal; pulse from 80 to 110; respiration from 20 to 25 or 30.

Autopsy. Tuberculous meningitis; miliary tuberculosis of both lungs; congestion of lungs.

CASE V.—*Abscess of Left Temporal Lobe in Left-handed Person.* A case under the care of Dr. Daniel M. Hoyt, from whom the earlier notes were obtained.

The patient, a man, had had scarlet fever when a child. In 1896 he caught a severe cold and discharge from both ears began soon after this. He noticed distinct loss of weight during that winter. In 1898 he had marked swelling and pain over the frontal sinuses. He had a section of bone removed from the nose about one month ago, and had had numerous operations for removal of nasal polyps. He has a continuous mucopurulent discharge from both ears.

May 22d. He complains of chilliness; pulse is 99; temperature 101°.

23d. He has pain over the left temple and is delirious.

Temperature 101°, evening 102°; pulse varies between 60 and 100. Pain over the temporosphenoidal region continues. Examination of the eye-grounds by Dr. Randall is negative.

26*th.* He has been more or less delirious since last note. Temperature is normal, pulse is 60. When he got up to pass urine gait was a little unsteady. Examination by nasal speculum shows pus coming from the upper portions of the nares, probably from the superior meatus. He is still in semi-stupor. The discharge from the left ear has notably lessened.

He was examined by me May 28th, and the following notes were made: He is partly stuporous and it is difficult to arouse him, but he can be aroused and replies correctly to simple questions. He recognizes me and calls me by name. He is not motor aphasic and not word-deaf. The irides respond to light. Pupillary reaction in accommodation and convergence cannot be determined because of difficulty in fixing the patient's attention. Extraocular muscles are normal. The muscular supply of each facial nerve is normal, the patient wrinkles his forehead, closes his eyelids, and draws up each corner of the mouth normally. The tongue is protruded straight and is normal. Each masseter muscle contracts well. Sensations for touch and pain are normal in the face and all the limbs.

The movements of the upper limbs are normal. The grasp of the hands is fair and equal on the two sides. The finger to the nose test shows no ataxia on either side.

The movements of the lower limbs are free at all parts. The patellar reflex is present on each side and probably diminished. He has no ankle clonus and no rigidity of the limbs. Sensitiveness of the sole of each foot is excessive, and therefore it is impossible to determine the presence of a Babinski reflex. Heel to knee test is fairly well performed on each side. He is not made to stand up as his condition does not permit the test. There are no localizing signs, and although the patient is not word-deaf he is left-handed, as is his child also, although he writes with his right hand.

Dr. Randall, thinking the source of infection might be in the nasal sinuses, following this examination, cleaned out the sphenoidal and ethmoidal cells, but found very little pus in these cavities. Pain over the left temporal region continues.

I examined him again May 29th and found that the stupor had increased and that he was no longer able to pay attention to what was said to him, and that it was impossible to arouse him. Babinski's reflex was now distinct and typical on both sides.

Dr. C. H. Frazier operated on May 29th. When the dura was opened a portion of the temporosphenoidal lobe protruded, showing considerable pressure. The handle of the knife was introduced between the dura and the brain toward the base of the skull, and at once a gush of flocculent cerebrospinal fluid occurred. The evacuation of this fluid at once relieved the excessive pressure and the brain collapsed within the opening. On further examination an abscess was found in the temporosphenoidal lobe, containing possibly 3 to 4 drams of pus. The pus was evacuated and the cavity in the base of the brain thoroughly irrigated. Leukocytes on June 4th were 19,040. He died June 6th.

The necropsy was performed in the presence of Dr. Hoyt, and from him I have obtained the following notes: From the trephine opening in the temporal region was found protruding an intermixture of blood clot and broken-down brain substance. Two stitches still remained in the upper angle of the wound in the dura. On removing the skull-cap the dura was found slightly adherent to the skull, its appearance opaque, not thickened, veins markedly congested and dilated, containing clotted blood and injecting fluid. Before removing the brain there was noted on the lateral surface of the left frontal lobe a collection of pus without apparently any marked destruction of the brain substance beneath, and apparently of recent formation. On removing the brain the dura was found exceedingly adherent upon the left side, especially in the middle fossa. The left temporosphenoidal lobe was found to be exceedingly soft throughout, and in its centre was an abscess cavity containing quantities of necrotic material, but from superficial examination no fresh pus. It was impossible to trace any direct communication between the collection of pus on the left frontal lobe and the cavity in the temporosphenoidal lobe. Spreading over the base of the brain around the vessels of the circle of Willis, the pons and both lobes of the cerebellum, was a collection of yellowish white pus with apparently some distruction of the brain substance at the base. This purulent material extended down into the spinal canal as far as could be seen through the foramen magnum. On an examination of the base of the skull the following facts were determined:

Over the frontal sinuses the bone was apparently perfectly normal. There were no sinuses leading into the sphenoidal or ethmoidal cells. The sphenoidal cells on both sides were inspected; they both contained a small amount of gelatinous material, the right more than the left. The walls of these cells appeared normal. Upon the anterior portion of the petrous portion of the left temporal bone were found several openings, from one of which, the largest, when it was opened further, pus exuded. These openings communicated with the tympanic cavity.

UNILATERAL ASCENDING PARALYSIS AND UNILATERAL DESCENDING PARALYSIS.

THEIR CLINICAL VARIETIES AND THEIR PATHOLOGIC CAUSES.*

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In December, 1899, I presented a paper to the Philadelphia Neurological Society in which I called attention to a new clinical type which I designated as unilateral progressive ascending paralysis.[1] Somewhat similar cases have since been described under such terms as progressively developing hemiplegia, chronic progressive hemiplegia, and ascending unilateral paralysis. Unilateral ascending paralysis is probably the simplest and. therefore, the best designation for the symptom-complex.

Since the publication of the first contributions on this type of paralysis, the subject has attracted some attention, but not perhaps as much as its importance warrants. Probably not a few cases of progressively ascending hemiplegia or hemiparesis have failed of record because of the slowness of development of the affection and the overlooking of the essential feature of upward progression. Some cases have, no doubt, been passed by as lacking in interest, or as probable instances of focal disease not worthy of elaborate report.

Practical medicine is advanced by directing attention to clinical types or entities which are peculiar in their

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* From the Department of Neurology of the University of Pennsylvania.

[1] Jour. Nerv. and Men. Dis., April, 1900.

method of development, their symptomatology or their pathologic causes. One point which has been brought out during the brief history of the existence of progressively ascending hemiplegia is that unilaterality and upward progression of the paralysis are exhibited by affections differing in their pathology.

A close study of the recorded cases of unilateral ascending paralysis shows that this clinical type in a more or less pure form may occur, 1, as the result of primary degeneration of the pyramidal tracts to which may be added other degenerative lesions; 2, as the early stage of disseminated sclerosis; 3, as the form assumed by unilateral amyotrophic sclerosis; 4, as the order of progression in unilateral paralysis agitans; 5, as the expression of a focal lesion either cerebral or spinal; 6, as a clinical type in cerebrospinal syphilis; and as a peripheral or a hysterical affection.

In my description of the first case (rather of the first two cases of unilateral ascending paralysis, for brief account was given of a second case seen many years before the publication of the paper), I suggested that the disease probably represented a new clinical type due to slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neuron systems.

The cases which have been recorded by others as well as by myself, since attention was first called by me to the clinical type of ascending hemiplegia, even those cases which have been regarded as instances of pyramidal degeneration, have varied somewhat in their clinical features. In some, for example, spasticity has been present from an early period; in others it has been absent or has been but slightly marked. The reflexes, usually exaggerated, have in one or two instances been diminished or only moderately marked; the wasting usually present but moderate has varied in amount in different cases. As the literature of this affection is still meager, I shall take occasion to give a résumé of all the cases so far published, unless, indeed, some reported case has escaped my investigations.

Before doing this, however, I desire to direct attention to unilateral descending paralysis, which is to be regarded as simply the same type reversed. Unilateral ascending paralysis due to degeneration of the pyramidal tracts, or to this and some added condition of degeneration, is the more common form of progressively developing hemiplegia, but an affection essentially the same sometimes takes a reversed order so far as the limbs or as the face and limbs are concerned, giving us a unilateral descending paralysis or a progressively descending hemiplegia. Through the courtesy of two of my professional colleagues in Philadelphia, Dr. David Riesman and Dr. William Pickett, I shall have the opportunity to present in the concluding portion of this paper two cases of this descending type, the one an example of disseminated sclerosis taking at first the unilateral descending and systemic form, and the other probably an instance of nearly pure pyramidal degeneration.

That the suggestion of a new clinical type is sometimes fruitful in the development of diagnosis is illustrated by the brief history of progressively developing hemiplegia. The report of two or three cases of unilateral ascending paralysis of unusual form has called attention to the question of ascending and descending unilateral types of certain well-known diseases, such as disseminated sclerosis, paralysis agitans and amyotrophic lateral sclerosis. Variations in the symptomatology of these reported cases have also awakened interest as to the probably differing pathology of some of the cases recorded.

After summarizing in the briefest manner possible the cases of unilateral ascending paralysis hitherto published, and after some discussion of the different forms of disease of the nervous system which may present a progressively developing hemiplegia, ascending or descending, I shall record one new case of unilateral ascending paralysis and one new case of unilateral descending paralysis, describing also the case referred to me by Dr. Riesman, when considering the unilateral ascending and the unilateral descending types of disseminated sclerosis.

Case 1.—A man, aged 52, about two years before coming under observation,[1] began to show signs of weakness in the right lower extremity. Weakness in the arm appeared eighteen months after the impairment in the lower extremity was first noticed. This soon became more and more evident, and was accompanied by the tendency to carry the arm raised against the body and flexed at the elbow.

When first examined the paresis in the upper extremity although easily determined, had not nearly reached the degree of impairment observed in the leg. The right side of the face was also slightly paretic. Wasting was distinct in the right lower extremity, the measurements showing a difference of one and a half inches for the thighs and five-eighths of an inch for the calves. The various movements of the right leg were distinctly weaker than those of the left, but were nowhere absolutely abolished; similarly all the movements of the right arm were distinctly impaired. Faradic contractility was retained. The affected limbs were not spastic nor contracted. Careful examination showed retention of all forms of sensation. The tendon and muscle phenomena on the right side were all somewhat exaggerated. Knee-jerk was plus on the left side, but considerably more exaggerated on the right. Patellar clonus and slight ankle clonus were present on the right, but not on the left. The plantar reflex was normal on the left; but on the right, while the Babinski response was not present, downward flexion of the toes was distinctly less marked than on the left.

Case 2.—A woman, aged 43, first noticed weakness in her left leg while pregnant with her last child, three years before coming under observation.[1] A few months later the left arm became paretic. The reflexes were much increased on the affected side. The patient remained under observation for many months, the paresis of the leg and arm slowly increasing. Neither leg nor arm was contractured, and sensibility was preserved. The patient complained at times of pains in the limbs and twitching in the leg and arm.

This woman was under my care more than 20 years ago and had previously been a patient of the late Dr. E. C. Seguin, who believed that there was a cerebral lesion causing secondary changes in the crossed pyramidal fasiculus. The view taken by me was that the alterations in the pyramidal tracts were of the nature of a lateral sclerosis, but were primary and not secondary to any cerebral lesion. I have learned that the patient was alive about fifteen years after she was first seen by me and had become entirely unable to walk. Just how she was affected at this time was not ascertained, except that she had become triplegic, the affection having evidently passed over to the lower extremity on the side opposite to that on which it had started.

Case 3.—A man, aged 41, four years before coming under the notice of the reporter,[2] began to have a sense of weakness in the left lower limb in walking. This weakness gradually increased, and in about a year the left upper limb began also to be weak. The patient was positive that a year had elapsed after the impairment in power in the lower limb before he felt any weakness in the left upper extremity. He had never had any symptoms of an apoplectic seizure.

This showed that the movements of the left lower limb were very spastic, but not in the least ataxic. The toes of the left foot scraped the ground, and this foot was in-

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2. Spiller, William G.: Philadelphia Med. Jour., Feb. 9, 1901

verted in walking. The left knee-jerk was much more marked than the right, which was, however, prompt. Ankle clonus and the Babinski response were present on the left, but not on the right. Sensation was normal. The left upper limb was weaker than the right; it was also spastic, but the weakness and spasticity were less than in the lower extremity. The left upper limb was held slightly flexed at the elbow and against the body, but no contractures were present anywhere. Speech was normal, but the mouth could not be drawn up as well on the left side as on the right. The tongue went slightly to the left on protrusion. Ophthalmoscopic examination showed white atrophy of the left optic nerve. Wasting which was present was like that occurring in hemiplegia.

CASE 4.—A youth, aged 19, about four years before coming under observation[3] began to drag his right foot; it tended to cross in front of the left, and he wore off the sole of his right shoe at the toe. Two years later he had diplopia, which lasted for five months. The right leg at this time was somewhat smaller than the left. About three and a half years after the right foot first began to drag, which was about five months before coming under observation, weakness was noticed in the right arm.

Examination showed that when the patient walked the right leg was rigid and the toe dragged. The movements of the right leg could be performed, but were distinctly weaker than those of the left. The right leg and arm were both somewhat atrophied. The adductor pollicis muscle of the right hand was not so large or firm as that of the left. The muscles everywhere responded to the faradic current; the tendon reflexes on the right were all exaggerated, and a well marked Babinski reflex was present on this side. The reflexes on the left, while marked, were much less active than those on the right. Plantar stimulation caused slight extension of the great toe and flexion of the others. Right-sided facial paresis was present. The muscles of the right side of the throat were also paretic, and the voice had a distinct nasal twang. Nystagmus and paresis of the left inferior rectus, and pallor of the temporal halves of the discs were other symptoms. All forms of sensibility were everywhere preserved. The patient showed a slight Romberg symptom, and at times while standing or walking, a tendency to lose his balance. The case was regarded as one of disseminated sclerosis, beginning as unilateral ascending paralysis.

CASE 5.—A man, aged 60 at the time of his death, about eight years before had some paralysis of the right extremities.[4] At this time knee-jerk and biceps-jerk were increased on the affected side. Four years before his death hospital notes showed some loss of power in both the right leg and right arm; and all the muscle and tendon phenomena on the right side were exaggerated, patellar clonus and ankle clonus both being present; also the speech was slightly affected, and sensory impairment and astereognosis were absent.

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[3] Potts, Charles S.: Jour. Nerv. and Ment. Dis., xxxviii, No. 10, October, 1901.

[4] Mills, Charles K., and Spiller, William G.: Jour. Nerv. and Ment. Dis., July, 1903.

Notes made between two and three weeks before death showed that he was markedly paralyzed in the right leg, and to a less extent in the right arm; also that he was paralyzed in the left leg, but not as seriously as the right. The paralyzed limbs were spastic, and the tendon and muscle phenomena were all exaggerated. He had ankle clonus on both sides, and patellar clonus and the Babinski response on the right. Sensory phenomena were absent. The face was not paralyzed. Contractures and wasting were absent. During two or three years before his death he was treated for cardiovascular symptoms, including attacks of angina.

Postmortem showed advanced disease of the heart, vessels and kidneys, the case being an extreme instance of arteriosclerosis. Chronic pleurisy and tuberculosis with cavity formations were present. Microscopic examination showed intense and long standing degeneration of the right crossed and left direct pyramidal tracts, the degeneration extending into the pons, but not into the left cerebral peduncle; also comparatively recent degeneration of the left crossed and the right direct pyramidal tracts, traced by the method of Marchi into the lower part of the right internal capsule. No lesions, degenerative or focal, were found elsewhere in the brain or spinal cord.

Case 6.—A young woman, aged 18 at the time of first coming under observation,[5] was noticed to show slight clumsiness and awkwardness in using the right hand and foot shortly before the age of fourteen. Gradually the weakness of the right lower and upper extremities increased, this from description being more marked proportionately in the former than in the latter. When between seventeen and a half and eighteen years old a slight swelling of the right half of the thyroid was noticed.

Examination showed no abnormality of cutaneous and muscular sensibility nor of the special senses. Pupillary reactions, movements of the ocular muscles and the fundus oculi were all normal. The sphincters were also normal and the patient was mentally sound. The reporter gives in careful detail a description of the musculature and of the movements performed or incapable of proper performance on the right half of the body. Summarized, this indicated marked loss of power and atrophy in both the right upper and lower extremities, considerably greater in their distal than in their proximal portions. The affected limbs were neither spastic nor ataxic, but gave rather the appearance of flaccidity or flabbiness. The paralysis and atrophy were not localized to special groups of muscles. Marked quantitative change to the faradic current was present, being especially noticeable in the muscles which showed the greatest wasting, these being the hand muscles, the anterior tibial and the peroneal. All the deep reflexes were greatly exaggerated on the right side. The Babinski extensor response was typically present, but there was only an indication of ankle clonus. Some paresis of the muscles supplied by the seventh nerve and marked loss of power in the motor distribution of the fifth nerve were present.

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[5] Patrick, Hugh T.: Jour. Nerv. and Ment. Dis., xxx, August, 1903.

Case 7.—A woman, aged 50, was seen in consultation in October, 1905.[6] In August, 1904, she first discovered some weakness of the right lower extremity, although it may have been present in mild degree before. The weakness and awkwardness in using the limb very gradually increased until about one or two months before coming under observation, since which time the impairment had increased more rapidly. The patient had suffered no pain in the extremity, in the back or in the head, and had no symptoms of cerebral disease. She had occasional headaches which seemed to be of the migraine type, and now and then had a slight vertigo, but this was probably due either to the condition of her stomach or to some arteriosclerosis.

Examination of the right lower extremity showed general paresis of moderate degree in the entire limb; that is, all movements were impaired in force without definite localized palsy of any group of muscles. The movements of flexion, extension, abduction and adduction of the foot and also intermediate movements were distinctly impaired as compared with those of the left foot, but the reduction in power extended to the entire limb, although it appeared to be more marked distally. The right knee-jerk was greatly exaggerated and patellar clonus could be elicited. Persistent ankle clonus and also a decided Babinski response were present on the right. Front tap was present and marked on the same side and the muscle jerks were also plus on this side. On the left the knee-jerk was somewhat above the usual norm, as were also the muscle jerks, but patellar clonus and front tap were absent. The plantar response on this side was irregular. Careful examination was made for sensory disorders, with absolutely negative results. Examinations seemed to show some slight impairment of the upper extremity, but so slight that it was hardly fair to record it as present.

Case 8.—A young man, aged 23, complained that for six months his left leg had been growing gradually weaker. He would sometimes trip because he could not clear the ground well with the tip of his left foot. At times also he could not use the left arm well. His condition for about one year is described by the reporter.[7]

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6. Mills, Charles K.: Jour. Nerv. and Ment. Dis., xxxiii, No. 2, February, 1906.

His walk suggested a foot drop on the left side, but did not show the circumduction frequently exhibited by hemiplegics. Dorsal flexion of the foot was very feeble, movements of the toes being almost completely lost. Movements at the left knee and hip could all be performed, but with less force than on the right. Knee-jerks and heel-jerks were lively on both sides, but much more marked on the left than on the right, and after three or four months ankle clonus could be elicited. The Babinski response was absent on both sides. The entire left lower extremity was not spastic but flaccid and the seat of general wasting. The left upper extremity was smaller than the right. Atrophy was also distinctly shown in the neck and shoulder. As time progressed the left upper extremity became completely paralyzed. The left face showed no paralysis or atrophy.

Examinations made several years after the first observation of the patient gave, by the Oppenheim method, a flexor response in the smaller toes of the right foot, and a distinct extensor response in the big toe of the left foot. The response to both the faradic and galvanic currents was normal. All forms of sensibility, the special senses, and the bladder and bowels were normal. Absence of exaggeration of the deep reflexes in both upper extremities was an incongruous feature of the case.

These cases represent the literature of this subject as known to me up to the time of the preparation of this paper. They all seem to present in their symptomatology evidences of the probable degeneration of the pyramidal tracts, crossed and direct. I shall recur again to Case 1, when discussing the subject of the unilateral ascending type of paralysis agitans, which this case was believed to be by Patrick. Whatever the case was in its totality, it certainly presented some of the features of a case of pyramidal degeneration. Case 2, seen by me more than 20 years ago, and, therefore, in reality, the first case observed, was without doubt one of progressively ascending hemiplegia due to pyramidal degeneration. Cases 3, 5, 6, 7 and 8 seem to belong to the type first indicated by me, although differing in some features, especially as regards spasticity, the reflexes and wasting. The solution of these differences can only be had as we gain more knowledge by the study of cases, and especially as we acquire additional pathologic data. Case 5, that of a patient originally observed in my wards at the Philadelphia General Hospital, and published by me in conjunction with Dr. Spiller, is the only one as yet in which necropsy and microscopic examin-

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7. Newmark, L.: Jour. Nerv. and Ment. Dis., xxxiii, No. 3, March, 1906.

ation have shown degeneration limited or almost limited to the crossed and direct pyramidal tracts. Case 4, recorded by Potts, was, in all probability, as he suggests, one taking the form of unilateral ascending paralysis in a comparatively early stage of disseminated sclerosis. This case will be presently referred to again when considering the unilateral ascending type of disseminated sclerosis.

I shall next present the details of an unrecorded case of unilateral ascending paralysis—one which does not seem to belong clearly with any one of the 8 cases just summarized. It is apparently an illustration of cerebrospinal syphilis, taking the unilateral ascending form.

Case 9.—The patient, a man, aged 55, had no family history of importance. He had worked in lead 10 years before coming under observation. Four years before this time his right leg began to grow weak; the impairment of power steadily increasing. The veins of the right leg were markedly varicose. A dull aching pain developed in the heel and in the calf in the region of the varicose veins. The aching had grown worse with the weakness. Three years after the onset he first began to notice weakness and aching in the right arm, these increasing steadily. Shortly after this weakness became noticeable in the left knee and ankle, with some ache in the heel. The left arm remained normal. The bladder and bowels were not involved. He occasionally suffered from backache.

Examination showed that in walking the patient dragged his right leg. Power in the right leg was greatly diminished but normal in the left. The right arm was weaker than the left. The right lower face was paretic. He could not draw the right angle of the mouth up; on smiling the left angle of the mouth was drawn upward more than the right. He could wink each eye separately, the left better than the right. Patient stated that his left arm and leg were beginning to feel as the right extremities felt a year or more before. Muscular atrophy was absent. Standing with his eyes shut he almost fell. Both knee-jerks were exaggerated, the right more than the left. The ankle-jerk was exaggerated on the right but diminished on the left. The Babinski response was obtained on the right, but not on the left. Biceps and triceps reflexes were increased on the right and normal on the left. Sensation for touch and pain was normal in the extremities and in the face. Speech was normal.

Eye examination revealed extensive chorioretinitis with well advanced optic nerve atrophy. The pupils responded sluggishly to light and more promptly to convergence. No diplopia or muscle insufficiency was present. The fields were much contracted, especially on the temporal side. Patient passed from under observation before an entirely satisfactory investigation of his case was made.

UNILATERAL DESCENDING PARALYSIS.

I had the opportunity of seeing and studying the following interesting case through the courtesy of Dr. William C. Pickett, of Philadelphia, whom I had requested, with others, to be on the lookout for cases of unilateral ascending and unilateral descending paralysis. Dr. Pickett, on first seeing the case, at once recognized that it belonged to the type of progressively developing hemiplegia, in this case taking a descending course.

Case 10.—A woman, about 45, had been well until three years before coming under observation, when for the first time she noticed a weakness in the left arm on attempting to use it. She soon lost the power of lifting and projecting the arm. The impairment of power became more marked in the distal than in the proximal portion of the limb, and a few months after the onset of the paresis the movements of the hand at the wrist and those of the fingers became much impaired.

About one year from the time when the arm was first affected she noticed some weakness in the left leg. In describing the condition of her lower extremity at this time she said that she felt as if she made miscalculations in stepping up or down. In walking, the toes and fore part of the foot would catch or not be lifted properly, causing her to stumble. She soon had a limping or dragging gait. In the course of five or six months a marked degree of paralysis developed in the left lower extremity. The left upper and lower extremities had been in a condition of gradually increasing helplessness during the last 18 months. The patient said that she felt as if she had to carry them with the other part of the body. She had also a curious feeling of loss of power in the abdomen or bowels of the left side. Her face and ocular muscles had not been affected.

Neither before nor since the coming on of the paralysis the patient had not had any severe headache, paroxysmal or continuous, or any spells of nausea, vertigo or vomiting. She had also had, so far as she knew, no disorder of sight, hearing, smell or taste. She had had no loss of control or other affection of the bladder. She had had no double vision or no difficulty in dragging in the affected limbs. Her menses had continued regular, and her bowels had also been regular with the exception of occasional constipation.

Careful examination gave the following results: Left arm was limp or flaccid, the only place where any contractures were shown was in the fingers, which were half flexed at the first and second phalangeal joints, but this condition of contracture was a passive rather than an active one. The bend of the fingers was evidently due rather to loss of power in the extensors than to any active spastic condition of the flexors. The fingers and thumb dropped into the hand. The loss of power in the limb was greater distally than proximally. She could elevate the entire extremity to an angle of only about 45 to 50 degrees with the shoulder. She could, however, bend the forearm about half way, and extend the arm against resistance, but with about half the normal power; in other words, the movements of the upper arm were considerably impaired but all retained. She could not pronate or supinate the forearm; she could not flex the hand on the wrists; she could not ulnar flex it or fully extend it, but had some power of extension, especially showing itself toward the radial side of the hand. She could not extend the fingers at the metacarpophalangeal articulations or distally. She had some slight flexor power in the movements of the fingers. She could adduct and abduct the thumb which lay helpless toward the palm. Inspection showed some apparent slight wasting of the suprascapular muscles and perhaps of the small muscles of the hand. There was not, however, the distinct atrophy which is so marked in amyotrophic lateral sclerosis and progressive muscular atrophy of the Aran Duchenne type.

MEASUREMENTS OF THE UPPER EXTREMITIES.

Middle of left forearm..	7⅜ in.	Middle of right forearm..	7⅝ in.
Left hand	7 in.	Right hand	7½ in.

She dragged the left leg in walking, but not with the circumduction movement of the foot which is shown in so many cases of hemiplegia. The lower extremity, like the upper, was partially paralyzed, but not in any part quite as markedly affected as the upper extremity. The loss of power was more marked distally than proximally. The movements of the left foot were tested and compared with those of the right, and all were found greatly impaired but none completely lost. Those most affected were dorsal flexion of the foot and dorsal flexion with adduction. The arch of the foot was somewhat flattened; the foot showed a tendency to turn outwards, exhibiting a partial valgus. The movements of the leg on the thigh, of the thigh on the pelvis and all other movements of the thigh could be performed with considerable force, but less energetically than the corresponding movements on the right side. The muscles of both the lower and upper extremities responded everywhere to the faradic current, but with some quantitative diminution, particularly in the upper limb.

MEASUREMENTS OF THE LOWER EXTREMITIES.

Middle of left leg......	11½ in.	Middle of right leg....	12⅛ in
Left foot	7¼ in.	Right foot	7½ in.

The two sides of the face were symmetrical. She had about equal control over the movements of the left and of the right side of the face. The tongue was protruded straight and showed no fibrillary tremor. No loss of power was present in the masseter, temporal and pterygoid muscles. Ocular movements were normal; there was no nystagmus. Articulation, enunciation, phonation and swallowing were normally performed. The pose of the body and head and the facial expression had none of the appearances seen in paralysis agitans. Knee-jerk and quadriceps jerk were much exaggerated on the left; they were present but not increased on the right. Patellar clonus was absent on both sides, as was also ankle clonus, although a slight tendency to clonus was twice shown on the left. Achilles jerk was marked on each side. A typical Babinski reflex was present on the left, the great toe extending distinctly and somewhat deliberately, the other toes more quickly but less decidedly. On the right the small toes were plantar flexed on stimulation of the sole, the great toe sometimes extended and sometimes flexed, but when it extended it was not in the same typical and deliberate manner as on the other side. Examination of the upper extremities was made for the wrist, and elbow jerks and the von Bechterew reflex and its modifications. The jerks were a little more marked on the left than on the right; the most marked responses were the triceps and the infra-spinatus which, while not greatly exaggerated, were undoubtedly more marked on the left than on the right side.

Well marked anesthesia to touch, pain and temperature was nowhere present, but careful and repeated examinations showed slight hypesthesia on most of the left half of the body like that so often seen in hysteria and hystero-neurasthenia of traumatic origin. Her answers, however, were not uniform as to the limitations of this slight hypesthesia. Above the waist, for instance, her replies seemed to show that the sensation was fully retained nearly all over the region of the left scapula and of the deltoid.

Dr. G. E. deSchweinitz made the following report: Vision, right eye 6/5; left eye, 6/5. A., right eye, 6.5 D; left eye, 6.5 D. Pupils normal in all their reactions; equal in size. No diplopia; no limitation of rotations of exterior ocular muscles. Esophoria less than one degree. Optic discs of good color; retinal circulation normal in distribution and carrying normally tinted blood; slight enlargement of retinal veins, but no decided disproportion between size of veins and arteries. No ophthalmoscopic changes in the retina or choroid. Dr. deSchweinitz remarks that the ocular examination is practically negative; that the eyes are normal; that a slight contraction of the color fields found to be present is probably due to lack of concentration from retinal tire induced by long examination. This ocular examination is important as indicating the absence of a focal lesion like a cerebral tumor.

This interesting case, which I examined with great care, is an excellent illustration of a progressively developing descending hemiplegia in all probability due to primary degeneration of the pyramidal tracts, with possibly some slight involvement of other parts of the neuraxis. With the exception of the slight hypesthesia on the affected side, the case was purely motor, and had the usual diagnostic concomitants of a purely motor case, namely, abnormal increase of the deep and superficial reflexes. The hypesthesia, like the sensation of dragging of the limbs, seemed to be due to suggestion. The case in this respect reminded me of the idea long since advocated by Charcot, that the slight or moderate impairment of sensation sometimes observed in cases apparently due to a strictly motor cerebral lesion was sometimes best explained on the hypothesis of hysteria or suggestion. It undoubtedly belongs to the clinical group of unilateral descending paralysis, the pathology being the same as that which is present in at least some cases of unilateral ascending paralysis, the degenerative lesion, however, having developed in the upper part of the spinal cord and later in the lower. It is unlike the cases presently to be described, the one referred to me by Dr. Riesman, in which some of the symptoms seemed to point unerringly to a disseminated sclerosis beginning as a descending hemiplegia. The descending types of amyotrophic lateral sclerosis and of unilateral paralysis agitans could be easily excluded, the former chiefly by the absence of true spasticity, decided wasting, fibrillary tremors, and partial degeneration reactions; the latter by the absence of tremor, masked face and fixed pose and all the truly typical features of Parkinson's disease. Nystagmus, disorders of speech and other symptoms and signs of disseminated sclerosis were absent. The case was inexplicable on the theory of a focal lesion; syphilis, hysterical paralysis and peripheral disease could all be excluded.

UNILATERAL ASCENDING TYPE OF DISSEMINATED SCLEROSIS.

Disseminated sclerosis may first show itself as a spastic paralysis which, beginning in the one lower extremity, ascends in a greater or less time to the upper. It can not be doubted that Case 4, reported by Potts, is one of this kind, as in addition to unilaterality and progressive ascent, this patient showed several of the typical signs of disseminated sclerosis, as nystagmus, pallor of the temporal halves of the discs and some incoördination in standing and walking. In cases of this description it is altogether probable that the sclerosis at first largely attacks the pyramidal tracts and assumes an almost systemic form. This tendency, to a large degree of systematization, is sometimes observed in cases of bilaterally beginning and progressively increasing disseminated sclerosis.

A case of the hemiplegic form of disseminated sclerosis recorded by Thomas and Long[8] differs from the case of Potts in that the symptoms were not purely motor.

Case 11.—A man, aged 47, seven years before his death became paretic in his right leg, the impairment of power increasing and having added to it diminution of sensibility and incontinence of urine and feces. Some improvement in the symptoms in his right leg occurred, but two years before his death these gradually increased and numbness and paralysis in the right arm appeared. The year before his death he had almost complete paralysis in the right lower extremity with contractures, exaggerated deep reflexes on the affected side, including ankle clonus; also slight atrophy of the lower extremity. The right upper extremity was a little paretic, the reflexes at the wrist and elbow were exaggerated in both upper extremities, but more on the right. Marked anesthesia was present in the right lower extremity and the right half of the trunk, and a hypesthesia in the hand and forearm. The special senses were unaffected. He died of an attack of acute pleurisy.

Plaques of sclerosis which, from their position and extent, explained the characteristics of the symptomatology and progress of the cases were found in the cervical and thoracic cord.

UNILATERAL DESCENDING PARALYSIS IN DISSEMINATED SCLEROSIS.

In rare instances, a form of more or less slowly descending paralysis is observed, that is, of paralysis beginning in the arm and face and later involving the leg. This, of course, is known to occur somewhat frequently in paralysis agitans, although after the plastic rigidity with or without tremor has attacked one upper extremity it is perhaps just as likely to cross to the opposite leg as to pass to the leg of the same side. Dr. David Riesman, of Philadelphia, has furnished me with the notes of a case which appears, when closely analyzed, to be one of disseminated sclerosis in which the paralytic, spastic and tremulous phenomena were first most marked in the upper extremity. So far as the limbs were concerned, the affection was, up to the time when the last notes were made, unilateral in its distribution. Some question may arise in the consideration of this case as to whether it is one of disseminated sclerosis of unilateral and descending type or a form of slowly developing unilateral paralysis due to meningomyelitis or some other form of specific lesion. On the whole, however, it would seem to me that the diagnosis which fits the case best is that of disseminated sclerosis, in the stage when the record was made, of unilateral and descending form.

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[8] Thomas, A., and Long, E.: Compt. rend. hebd. des Seances de la Soc. de Biol., Oct. 13, 1899, No. 28.

The patient was a man forty-three years old whose father died of nephrolithiasis at the age of sixty-three and his mother of apoplexy at the age of fifty-eight. One brother was in an insane asylum for two periods of six months each.

Case 12.—Patient had primary specific lesion at the age of 26, accompanied by alopecia and later by secondary eruptions on the abdomen, thorax and arms. He took medicine for this disease for one year. He had pyelitis 8 years before coming under observation, suffering from this affection for one year. According to his statements, he is especially subject to erysipelas, which he had had about six times in all. Two of the attacks of erysipelas he had had within 18 months, the supraorbital and infraorbital regions being affected. The patient's most evident symptoms when he came to the hospital were partial loss of motor power in the right upper and lower extremity, accompanied by violent coarse tremors of the extremities when he attempted any movements of them. He limped somewhat with the right leg. He dated his present trouble to two years before he was seen, when he began to have a dull heavy feeling in the right shoulder. He then began to lose control of the right arm, which loss of power was accompanied by trembling of the arm when he attempted to use it. He stated that within the last year, that is, about a year after the onset of the paralysis and tremor in the arm, the right lower extremity had troubled him because of a loss of power in this part. He had had sharp shooting pains in this lower extremity. He had been able to walk in the dark without falling. Both bowel and bladder control had remained intact. He had never had any girdle sensation. Consciousness had never been lost.

This showed his speech to be somewhat monotonous, with a tendency toward scanning speech; spasticity of right leg, thigh and arm; marked intention tremor of the right upper extremity; fibrillary tremor of the tongue; some right-sided suprascapular atrophy; nystagmus; increased right knee-jerk with slight ankle clonus; normal reaction of pupils to light and distance; and intact sensation. Later notes made by Dr. Riesman showed that the left pupil was larger than the right; also that the pupils reacted sluggishly both to light and accommodation, the left not as well as the right. There was also no apparent contraction of the visual fields. The tremor in the right upper extremity was most noticeable when moving from a resting position and it seemed to disappear at times when the arms were supported. It was very noticeable when the arm was held free. The tremor was not rotary or rhythmic, but jerky and varying in amplitude. No pill-rolling motion like that seen in paralysis agitans was present. The patient tended to hold the fingers somewhat flexed at the knuckles. The hand, arm and shoulder girdle showed no atrophy. All movements of the upper extremity could be performed, but were affected by the tremor. Slight tremor was present in the right leg on its being moved, with no wasting of this extremity and no apparent shortening, although the patient said that the leg felt shorter than the other. Extension and flexion of the right foot were ample, but more sluggish than similar movements of the other foot. There was marked limitation of rotation of the right leg; other movements were normal. Knee-jerk was increased on the right side; slight tendency to ankle clonus was shown and a marked Babinski reflex was present on this side. Sensation was normal. The gait of the patient was hemiplegic, the right leg being dragged somewhat. Distinct right-sided ptosis is noted in one entry. The lungs, heart, blood, abdominal viscera and all parts of the body were carefully investigated, and notes of these examinations have been furnished me by Dr. Riesman, but as they seem to have no special bearing on the man's nervous disease they have been omitted for the sake of brevity.

Pupils 4 m.; irides react to light and accommodations; tension normal; in the right eye the media were clear and the disc pale, showing atrophic excavations; there was myopic astigmatism, 1 D. The left eye was the same as the right. Ocular movements were unimpaired. It is stated in the history that the patient had diplopia fifteen years before coming under notice, this having been corrected by glasses.

ASCENDING TYPE OF UNILATERAL AMYOTROPHIC LATERAL SCLEROSIS.

Unilateral amyotrophic lateral sclerosis, a comparatively rare form of amyotrophic disease, is occasionally ascending, although this is not the rule. Spiller[9] succeeded in collecting 10 cases of unilateral amyotrophic lateral sclerosis; of these, four were with necropsy. Three of the ten were of ascending type (Vierordt, Mott, Senator). To these is added a case of his own, with necropsy and microscopic examination.

The paralysis began in the right lower extremity, passed to the upper limb of the same side, and later to the left lower extremity, the left upper extremity being also eventually slightly involved. In this case, besides spastic paralysis and abnormally exaggerated reflexes, considerable atrophy was present in the right upper extremity. Degeneration in this case was found in the ventral horns as well as in the pyramidal tracts. It is probable that, at least, in some cases which begin with pyramidal tract degeneration, if the patient lives long enough the ventral horns eventually take part in the degeneration; although, as already shown in the case recorded by myself in collaboration with Dr. Spiller,[4] pathologic examination showed that the cells of the ventral cornua were not involved to any appreciable extent in this unique case.

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[9] Spiller, William G.: Univ. Penna. Med. Bull., January and February, 1905.

After the publication of the paper by Spiller, an additional case of amyotrophic lateral sclerosis, at first unilateral and descending, has been recorded by Potts.[10] Both sides of the body were eventually involved, bulbar symptoms appearing and becoming prominent.

DESCENDING TYPE OF UNILATERAL AMYOTROPHIC LATERAL SCLEROSIS.

Seven of the ten cases collected by Spiller were of descending type (Dejerine, Pick, Leyden, Lennmalm, 3 cases of Probst). Of these, five began with bulbar symptoms, the paralysis of the upper and lower extremities coming on in succession, or at least later. In a case reported by Dejerine, beginning with bulbar symptoms, the lower extremity of the side affected was more markedly paralyzed than the upper. In Pick's case paralysis began first in the left upper limb and extended to the lower of the same side; later the right side became affected. In Leyden's case bulbar symptoms were first; next weakness developed in the left upper limb and later in the left lower extremity. In Lennmalm's case bulbar symptoms were followed by loss of power in the extremities of the right side and later by paralysis in the left upper limb.

In one of the several cases reported by Probst bulbar symptoms began first, the right upper and lower extremities becoming involved. In another case reported by Probst, weakness began in the left upper and lower limbs and later in the right upper. In still another case of Probst's bulbar symptoms began first, followed by weakness in the left upper extremity and in a short time in the left lower extremity.

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[10] Potts, Charles S.: Univ. Penna. Med. Bull., July and August, 1905.

Recently, I had the opportunity of studying a case of unilateral amyotrophic lateral sclerosis of descending type which it may be worth while to record in this connection, owing to the fact which has just been shown that scarcely more than a dozen unilateral cases of this disease have as yet been put on record.

Case 13.—A boy, aged about 14, was in good health until about three years before he was seen by me for his family physician, when he began to have slight tremor in the right hand. About eighteen months to two years later some weakness in the right leg was observed.

Examination showed no particular difference in the two sides of the head and face. Ocular movements were normal, as were also movements of the parts supplied by both the facial and trigeminal nerves. The right upper extremity showed distinct and nearly generalized atrophy, and some wasting was present in the right half of the trunk. The right thenar and hypothenar eminences were extremely wasted. The right lower extremity, like the upper, was also generally smaller than the left. Testing for special movements, weakness was found in all parts of the limb, but this was much more decided in the proximal than in the distal portions. The movements of the foot on the leg and the movements of the toes were greatly impaired. Every possible movement of the foot and toes was tested, with the result just given. Farado-contractures were well preserved on both sides; on the right side was a quantitative increase, the muscles generally responding to a current of less current strength on the right than on the left side. A moderate current produced widespread contractures in the general musculature of the arm, probably partial degeneration reaction.

On attempting movements of the right or atrophied arm the patient had associated movements of the left. No matter what movement was attempted with the right upper extremity, the left upper extremity responded, sometimes in regular and sometimes in a sort of forced athetoid movement of the hand and fingers. Voluntary movements of the right lower extremity did not cause movement in the left lower, but did cause some movement in the left upper extremity. These, however, were by no means as marked as when the voluntary effort was made with the upper right extremity. Both the deep and superficial reflexes were everywhere greatly exaggerated on the right side, the muscle and tendon jerks of both arm and leg being carefully examined. The right great toe remained permanently somewhat extended as in a case of Friedreich's ataxia. A marked Babinski reflex was present on this side, the already extended great toe extending still further on plantar stimulation. Ankle clonus was present on the right side. The wasting and loss of power were not present anywhere in the left half of the body. The reflexes, while prompt, were not exaggerated as on the other side, and irritation of the sole of the foot gave normal plantar flexion. A very slight hypesthesia was present in the right arm and trunk. The patient's mental condition was good. He had kept up with his class in a good preparatory school. He had neither continuous nor paroxysmal headache nor any other evidences of focal disease. The case seemed to be clearly one of unilateral amyotrophic lateral sclerosis beginning first in the upper extremity.

UNILATERAL ASCENDING TYPE OF PARALYSIS AGITANS.

It is, of course, well known that paralysis agitans is often for a long time a unilateral affection; it is not so well understood that the unilateral form of this disease not infrequently is of ascending type, the disease passing from lower to upper extremity of the same side and later to the opposite lower and upper extremity. Patrick regarded one of the cases described by me in 1900 as an illustration of this affection rather than a case of primary degeneration of the pyramidal tracts, but of this I have not been convinced. With regard to this matter I need only reproduce here remarks made in a previous paper written in collaboration with Dr. William G. Spiller:[4]

> Among the reasons for believing that the case was not paralysis agitans were the absence of spontaneous tremor; the presence of markedly exaggerated reflexes on the affected side, even including ankle clonus and patellar clonus; the existence of decided wasting on one side, especially of the lower extremity; and the absence of the facies of paralysis agitans and of a fixed position of the body and head. According to Sir William Gowers[11] both primary lateral sclerosis and paralysis agitans are probably abiotrophies, and it may be that in the case under consideration the two abiotic diseases are conjoined.

Patrick[5] reports a case of unilateral paralysis agitans in his paper with the view of indicating the similarity of this case to that reported by me and reinvestigated by him. In this case the disease began in the upper extremity, passing later to the lower. My impression is that this is rather the usual course, although I have not made any collection of cases to demonstrate the point. Whether or not the case reported both by Patrick and myself was one of paralysis agitans or of paralysis agitans and pyramidal degeneration conjoint, it is certain that Parkinson's disease may remain for a considerable time unilateral, and beginning in the lower extremity may take an ascending course. Recently, I have seen two cases of this ascending type.

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[11] Gowers, Sir William: Lancet, April 12, 1902.

Case 14.—A man, aged 55, a pattern maker, first began to notice stiffness or impairment in movement in his left leg in December, 1904. At first and for some time there was no involvement of the left upper extremity, but this gradually came on, and at the time when he was first seen, about 16 months after the beginning of the trouble in the leg, the arm was very markedly affected. He had occasional cramps in the legs, the toes at times contracting when these cramp-like feelings were present. He also had at times, especially after sitting, feelings of numbness or tingling in the left leg and arm.

Examination showed a rather fixed position of the head, slowness of gait with some dragging of the left foot, and a marked tremor in the left upper extremity, especially in the hand, this being the type usually seen in Parkinson's disease. Observed over short periods it was sometimes present and at others absent, and it was a little increased by voluntary effort or attention. While both the leg and arm on the affected side showed a little stiffness and awkwardness in movement, real loss of power was slight. The patient still continued to work at his business, even using the left hand. He said that if he could get a good grip on an object with this hand he could go on using it in his work. Sensation was everywhere retained.

Case 15.—A woman, aged 43, first observed weakness in the left lower extremity four years before coming under observation. Tremor did not appear for three years in the left lower extremity and shortly after this the upper extremity became involved, showing both weakness and tremor. Symptoms appeared in the lower extremity of the opposite side three and a half years after the beginning of the disease. The case was clearly one of paralysis agitans and need not be described at length.

It is not my intention to go with any elaboration into a discussion of the differential diagnosis of unilateral ascending and unilateral descending paralysis from focal lesions, cerebral or spinal, from cerebrospinal syphilis, and from peripheral or hysterical affections. For the sake of completeness, however, a few words may be said about these diagnoses. In a rare case a brain tumor may originate in the cortical leg area or beneath it and develop toward the arm and face regions so as to cause a progressive hemiplegia of the ascending type. The reverse might, of course, take place, and the tumor originating in or near the face or arm area and progressing toward the leg center, give a unilateral descending paralysis. In such a case some or all of the general symptoms of brain tumor would be likely to be present, and other focal signs of this affection, especially Jacksonian epilepsy, would sooner or later appear. A tumor of the internal capsule would most probably produce loss of power in both arm and leg nearly simultaneously. In some cases of arteriosclerosis necrotic areas form first in one and then in another portion of the brain, and it might happen that these would be so placed in succession in the motor region as to give unilateral ascending or unilateral descending paralysis. Such a case would develop clinically by steps rather than by regular progression, and other signs or symptoms indicating the character of the lesions could probably be detected if sought for diligently. A single tumor or necrotic lesion of the spinal cord would not cause a paralysis ascending from the leg to the arm, as the cerebrospinal tracts passing to the nerve nuclei for the upper extremity do not proceed lower than the upper thoracic region. Peculiarly situated and peculiarly progressing focal lesions of the cervical cord might simulate a unilateral descending paralysis, but the other well-known symptoms of such a unilateral focal lesion would be present and the subject hardly needs discussion.

The forms of spinal and cerebrospinal syphilis are protean. A spinal or intracranial gumma or gummatous meningitis or a succession of focal syphilitic lesions might give rise to a one-sided paralysis appearing first in the leg and afterward in the arm, and later taking the form of a triplegia. This is, indeed, what occurs in some cases of meningitis or meningomyelitis with a secondary degeneration of the pyramidal tracts. The new case of unilateral ascending paralysis (Case 9), had some of the features of a cerebrospinal syphilitic affection, although the patient denied venereal disease. Close attention to the history, and the presence of other evidences of syphilis and the variable and irregular manner in which the paralytic symptoms appear will assist in differentiating.

A paralysis of peripheral origin, of ascending or descending type, is rare, but, of course, may occur. Such a case would probably depend either on the degenerative neuritis or on the succession of peripheral lesions. The manner in which each stage of the paralysis appeared, the state of the reflexes, the results of electrical examination, and other well-known methods of determining the existence of a peripheral nerve disorder would serve to separate such a neural case from the forms of progressive hemiplegia under consideration. The absence of hysterical stigmata, as well as the slowly progressive manner of appearance of the paralysis of leg, arm and face would make a hysterical hemiplegia improbable. A pure neurasthenia never shows itself as a slowly developed and permanent hemiplegia.

A PATHOLOGIC STUDY OF SEVEN CASES OF PARALYSIS WITHOUT GROSS ANATOMIC CHANGE

IN RELATION TO THE CAUSE OR CAUSES OF UREMIC HEMIPLEGIA.*

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The following report embodies the results of a pathologic study of seven cases of paralysis associated with chronic kidney disease, such as are not infrequently regarded as of uremic origin, in which no gross anatomic change was found to account for the paralysis. These cases were studied with a view of determining, if possible, the cause, or causes, of paralysis, as we observe it, in persons brought to the hospital who lived under observation but a short time, and who present paralysis which can not be attributed to cerebral hemorrhage, with which the evidences of grave nephritis are often associated.

In Cases 1, 2, 3, 4, 5 and 7, the findings warranted a diagnosis of cerebral syphilis, and in Case 6 the lesion, although not entirely characteristic of syphilis, pointed to a similar diagnosis. In this case there was much arterial change, and the perivascular spaces of the paracentral cortex were markedly distended. In Cases 4 and 2 microscopic areas of softening were found in the cortex. In one section from the paracentral region in Case 4 only one of these areas was observed. In a section from the paracentral region from Case 2 two small areas were found in one field. In all probability these cases were of syphilitic origin.

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The microscopic areas of softening just referred to have an important bearing in explaining cases of paralysis in which no gross lesion is observable. Even in a microscopic study of the nervous system they could readily be overlooked. Though these areas are very small, perhaps only the size of a point of a pin, it is conceivable that if numerous, they could bring about some distinct alterations in the functions of the cortical nervous tissue, and sufficient indeed to cause paralysis. It seems highly probable that this lesion is responsible for at least some cases (what proportion it is impossible to state) of so-called uremic hemiplegia.

In a previous communication[1] I described these necrotic foci in connection with double hemiplegia. They were present in the paracentral region, and were associated with a disease of the wall of the blood vessels, round cell infiltration of the pia, and other syphilitic lesions. While it is well known that areas of softening occur in various portions of the brain, they are usually extensive enough to be recognized by the naked eye. More interesting and important are the minute areas of necrosis which are visible only under the microscope. These findings must modify the views previously held about uremic hemiplegia and other forms of hemiplegia in which there is no gross cause demonstrable at the autopsy.

Several theories have been offered to explain the paralysis occurring during uremia, but convincing pathologic evidence is lacking to a great extent. In few of the cases reported careful histologic studies have been made, and the reports of cases without such studies are valueless as evidence.

The mechanical theory, which explains the paralysis of uremia as due to cerebral edema, was first advanced by Traube. Raymond,[2] Tenneson,[3] Chantemesse,[3] Leichtenstern,[4] and others found cerebral edema at the autopsy. Le Corche[4] and Tallamande[4] claimed that the edema is sometimes localized, a view supported by

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[1] Am. Jour. Med. Sci., May, 1906.

[2] Rev. de Med., 1895, p. 705.

[3] Rev. de Med., 1885, p. 935.

[4] Article by Le Calvé : Arch. gen. de Med., 1903, p. 577.

Pätsch[5] and Charpentier.[6] Le Calvé[4] believed that the paralysis is caused by a fugacious edema.

The toxic theory, which holds that the cerebrospinal fluid is toxic and elective for the nerve cells, was advanced by Bouchard,[7] and supported by Landois,[4] Lanbuscher[4] and Ziehen.[4] Castaigne[8] claims to have demonstrated by certain experiments that the cerebrospinal fluid taken from patients suffering from nervous uremia contained elements which were toxic for nerve cells. He injected into the cerebral hemisphere of guinea-pigs fluid obtained by lumbar puncture in cases of nervous uremia. Four pigs thus treated with 1¼ or 1 cubic centimeter of this fluid, died in convulsions in one to ten hours. The researches of Leane, Sicarde and Widal are quoted by Castaigne[8] as showing that normal

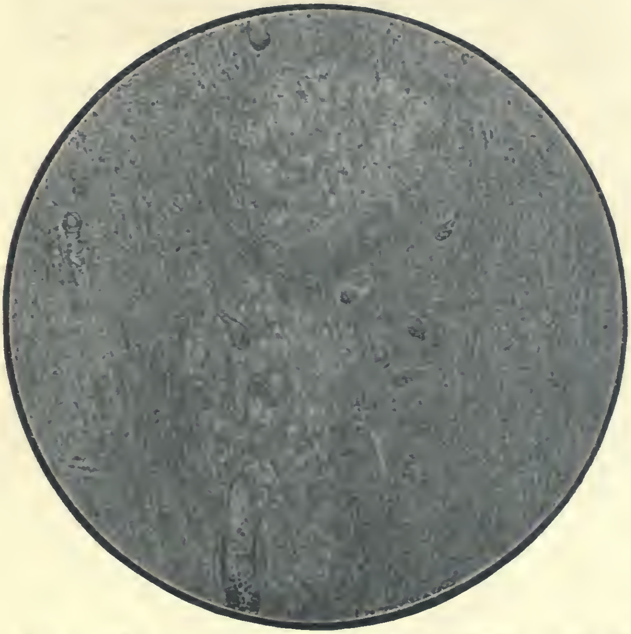

Fig. 1.—Case 2. Two microscopic areas of softening in the left paracentral region.

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[6] Arch. gen. de Med., 1887, p. 1.
[5] Ztschr. f. klin. Med., 1881, p. 209.
[7] Jour. des practicien., 1904, p. 131.
[8] Comp. rend. de la Soc. de Biol., 1900, p. 907.

cerebrospinal fluid injected in a similar manner was not toxic.

It is well-known that many cases of uremic hemiplegia have been reported without demonstrable lesions. Baillet[9] examined 37 cases and found no lesion in 29. This fact has led to the conclusion by some that the paralysis is due to autointoxication. Such was the conclusion of Brodier,[9] who reported 2 cases.

The paralysis of uremia has been attributed to arteriosclerosis by some observers, and this is in some cases undoubtedly the cause.

D. J. McCarthy[10] believes that many of the cases diagnosed as uremic hemiplegia, in which the autopsy shows extensive cerebral arteriosclerosis, the paralyses are not the result of a toxemia of the cortex, but rather of a malnutrition of the cortical cells due to the obliteration of the vessels supplying the cortex. The nerve cells under these conditions being badly supplied with nourishment on account of the deficient circulation, may become temporarily exhausted and thus produce, when situated in the motor areas, a temporary hemiplegia.

In a number of cases reported by Raymond[2] atheroma of the cerebral arteries was present. Raymond believed that uremic paralysis is due to an edematous state of the brain substance, which either awakens an old lesion, or produces certain vascular phenomena. The edema causes, in his opinion, at the site of the capillaries, a sufficient constraint in the circulation, in conjunction with the anemia which is always present in kidney disease, to bring about apoplectic attacks. His experiments in this connection are interesting and worthy of quotation.

Experiment 1.—He removed the left superior cervical ganglion of a rabbit and on the following day ligated the ureters. Four days later convulsions appeared on the right side of the rabbit's body. At the autopsy a notable quantity of fluid was found in the ventricles, and a slight degree of edema which was of a uniform degree on both sides.

Experiment 2.—In a second rabbit he removed the right cervical ganglion and six days later tied the ureters. In 4 days convulsions occurred on the left side of the body. In other words, convulsions appeared on that side of the body controlled by that half of the brain which had been deprived of the influence of its vasomotor nerves.

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[9] Article by Brodier: Arch. gen. de Med., 1900, p. 475.

[10] Proc. Coll. Phys. Phila., 1904, p. 248.

Experiment 3.—This experiment consisted of trephining a dog and introducing into the cortical region a small crystal of silver nitrate. The animal was paralyzed in the right leg on the following day. A month later the paralysis had completely disappeared. The jugular vein was then ligated on the left side, when shortly the paralysis of the right leg became as marked as on the day of the primary experiment. From this the animal finally recovered, and at autopsy 2½ months later an area of softening was observed at the site of the operation, and there was slightly more congestion of the left side of the brain than on the right.

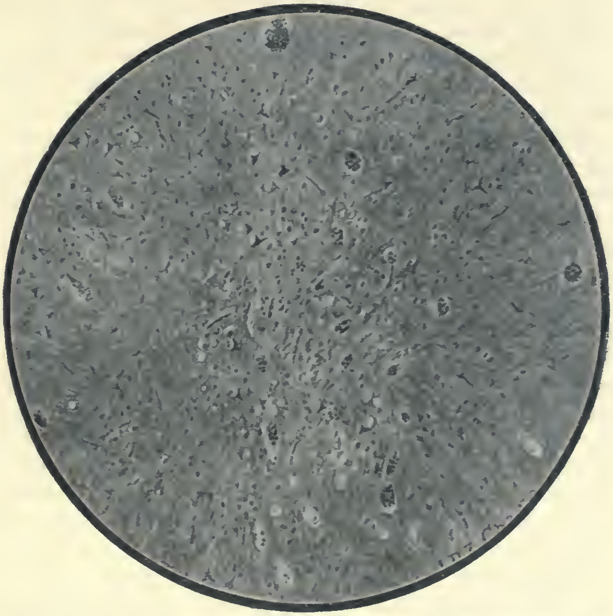

Fig. 2.—Case 4. One microscopic area of softening in right paracentral region.

Experiment 4.—This experiment consisted in trephining a dog and injuring part of the cortex on the left side by means of a thermocautery. Paralysis of the right posterior leg followed, gradually disappearing after 30 days. At the end of a month the jugular vein was ligated on the left side. Following this operation there was a recurrence of slight paralysis of the right leg which diminished at the end of a month and finally disappeared. At autopsy a lesion of the cortex was observed, but no difference between the two hemispheres from the point of view of the state of the circulation.

These experiments, he claimed, demonstrate that circulatory disturbances have an influence on the reappearance of old paralyses of cortical origin.

Weisenberg[11] concluded from the study of a case diagnosed as uremic hemiplegia that changes may be produced in the nerve cells of the brain and spinal cord by toxic processes like uremia. He has demonstrated acute degeneration of the motor filters of the brain and cord in association with cellular degeneration of the cortex.

In a few autopsies actual lesions have been demonstrated. Fürstner[12] reported a case with a history that five months before coming under observation the patient suffered from two attacks of right-sided paresis following the uremic state. At the autopsy two small foci of softening in the corona radiata and in the pons were found, and in this case he also demonstrated degeneration of both crossed pyramidal tracts and one direct pyramidal tract. In the case reported by Raymond[2] an old hemorrhagic focus was found in the right lenticular nucleus. No secondary degeneration was observed.

The changes in the nerve cells have been studied with a view to throwing some light on this subject. Lugaro[13] found in a case of uremia, with symptoms simulating tumor of the frontal lobe, chromatolysis and pigmentation of the cortical cells. Ewing[14] examined the central nervous system in two cases, and the cord in three cases of eclampsia, but was unable to find any peculiar cellular lesion excepting some nuclear changes of the Purkinje cells in one case. He reports also the cellular lesions found in six cases of uremia, in only one of which was there paralysis. He was able to conclude that uremia in the human subject is associated with change in the chromatic substance of the nerve cells. The spinal cells do not show any change as a rule, the greatest change appearing in the medullary nuclei, especially in the tenth nuclei. Ewing[14] quotes the experiments of Acquisto, Sacerdotti, Cerletti and Sambalino. Acquisto[14] and Pusateri[14] found different stages of chromatolysis in the cortical cells of the brains of dogs suffering from experimental uremia. Sacerdotti[14] and Ottolenghi[14] did not confirm these findings. The results obtained by Donetti[15] closely correspond to those described by Acquisto and Pusateri. Ewing[14] quotes also the experiments of Gabbi, who demonstrated a perinuclear chromatolysis with a homogeneous condition of the nuclei in the cells of the cerebral cortex of guinea-pigs and rabbits after bilateral ligation of the ureters. Marinesco is quoted by Chauffard[16] as having discovered chromatolysis of the nerve cells, and alteration of the nuclei by the Nissl method in uremia. The original reference could not be found.[17]

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[11] Jour. Nerv. and Ment. Dis., July, 1904.

[12] Neurol. Centrbl., 1898, p. 607.

[13] Rev. Neurol., 1900, p. 669.

[14] Article by Ewing: Arch. of Jour. Neurol. and Psycho-Pathol., 1898, p. 351.

Notwithstanding all these studies the cause of uremic hemiplegia has not been satisfactorily explained. One is forced to the conclusion that it may be due not to any one cause in all cases, but to one of a number of causes; in some cases, perhaps, to autointoxication; in others to edema or arteriosclerosis; and in still others to the presence of microscopic areas of necrosis which have been described in this paper.

Some confusion seems to exist as to what really constitutes uremic hemiplegia. It is a mistake to diagnose all cases of one-sided paralysis occurring in a subject who suffers from chronic kidney disease, uremic paralysis. Strictly speaking, a diagnosis of uremic paralysis should be restricted to those cases which develop during uremia, transient palsy, or a palsy which may persist to the end in fatal cases. There is no way, however, that I know of, by which it can be foretold whether the paralysis is of syphilitic origin, or is due to arteriosclerosis, autointoxication, or to cerebral edema.

In making a diagnosis of uremic paralysis we must search for the signs of intoxication of renal origin, such as general edema, pulmonary edema; premonitory symptoms, such as vertigo and gastric disturbances; the presence of albumin and casts in the urine, and other well-known symptoms of uremia. The diagnosis in old people is more difficult on account of the resemblance to symptoms following hemorrhage and softening of the brain, affections which are common in the aged. The variability and transitory character of the paralysis should always make one think of uremia as its cause.

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[15] Comp. rend. de la Soc. Biol., 1897, p. 502.

[16] La Bull. Med., 1902, p. 203.

[17] For further literature the reader is referred to: Lancereaux: L'Union Med., 1887, p. 705; and Colleville: Gaz. Hebdom., 1900, p. 673.

CASE REPORTS.

In none of the following cases was any gross lesion of the brain or cord demonstrable at the autopsy.

Case 1.—W. N., aged 68, a minstrel by occupation, was admitted to the hospital, in the service of Dr. Charles K. Mills, September, 1905. Except for the fact that his father died as a result of an attack of apoplexy, the family and previous histories were entirely negative. He dated his condition on admission, from an accident which occurred two years before, though it is doubtful that this had much bearing on his condition when admitted. While trying to board a car he was knocked down, striking his right hip on the car step. He was unable to get up or to walk, and his urine was passed involuntarily.

Examination.—On examination, after his admission, it was found that both grips were weak, the left decidedly so. The gait was tottering, although he was able to move both legs well. The tongue was protruded in the median line. There was no paralysis of the external ocular muscles, and apparently no paralysis of the cranial nerves. The pupils were moderately dilated, and his mentality seemed good. The station was fairly good with the eyes closed. Both knee-jerks were quick and slightly spastic. On stroking the plantar surface of the feet the toes were flexed. The radial and temporal vessels were distinctly atheromatous. Examination of the abdominal and thoracic viscera was practically negative. The specific gravity of the urine was 1,002 to 1,016; there was a trace of albumin, hyalin casts, leucocytes and epithelial cells. The patient died Dec. 9, 1905.

Autopsy.—Hypertrophy of the left ventricle was found, with general arteriosclerosis and interstitial nephritis. There was no gross lesion in the brain and cord.

Sections from the paracentral regions and the medulla oblongata were stained by hemalum, Weigert hematoxylin and thionin stains. The pia was moderately infiltrated with round cells. The blood vessels were distinctly thickened in the left paracentral region. This change was slightly more marked in the right paracentral region. No change could be demonstrated in the nerve cells when stained with thionin. There was a slight old degeneration of the pyramid on the right side of the medulla oblongata.

This case was not studied further as it was probably one of cerebral syphilis, and showed an old degeneration of the pyramidal tract.

Case 2.—W. L., aged 61, was admitted to the hospital in the service of Dr. Mills, Nov. 27, 1905. There was nothing worthy of mention in the family or previous histories.

Examination.—Patient was a somewhat emaciated old woman who was very dull of intellect and talked incoherently.

She did not understand what was said to her, although she could recognize persons. The left arm was spastic and contractures were beginning to make their appearance. The grip of the left hand was very weak. The left lower extremity was held rigidly in an extended position, and there was some atrophy present. Contractures were beginning to make their appearance here also, and the foot was held rigidly. There was a marked contraction of the Achilles tendon, with a dorsal flexion of the great toe. Power and motion in the right arm were good, but there was apparently some weakness in the right leg. The face was slightly drawn to the right, and the nasolabial fold on the right was more distinct than on the left. The tongue was protruded slightly to the right, although this may have been due to the absence of teeth on that side. Stroking the plantar surface of the foot was followed by marked plantar flexion on the right and dorsal flexion on the left. The knee-jerk on the right was increased, and also on the left side, although not to the same degree. The Achilles jerk was absent on both sides. There was no ankle clonus on either side. Examination of the urine showed a specific gravity of 1,016, a trace of albumin, and microscopically numerous leucocytes and epithelial cells, but no casts. Patient died Dec. 18, 1905.

Autopsy.—Autopsy was performed the next day. The pathologic diagnosis was croupous pneumonia of both lobes, emphysema, mitral stenosis and parenchymatous nephritis.

Microscopic Findings.—Sections from the right and left paracentral regions, and from the peduncles were studied microscopically, by the Marchi and Weigert hematoxylin methods, and stained by eosin, hemalum and thionin. The pia in the left paracentral region showed a moderate amount of round cell infiltration. In the cortex of the left paracentral region two small areas were observed, evidently beginning softening. These areas were very small, but were undoubted areas of softening, as the compound granular cells were distinctly observed in places. None of these areas was observed in the right paracentral cortex, but here a moderate degree of perivascular distension was present. In the right foot of the peduncle, near the inner portion, or corresponding to the second fourth from within outward there was an acute degeneration by the Marchi method. Cells stained by Nissl's method did not appear to be abnormal. This was probably a case of syphilitic disease. The significance of these small areas of softening already referred to should not be underestimated.

Case 3.—H. G., aged 49, was admitted to the hospital in the service of Dr. William G. Spiller, April 15, 1905. The only facts in the family and previous histories which need be mentioned are the fact that her mother died of a "stroke" at the age of 71, and that the patient had two stillborn children and one miscarriage. A year previous to her admission to the hospital she was seized with a dizziness which made her stagger "like a drunken person," but at no time to fall. She had also had some occipital headache. Five months after admission she was forced to take to her bed, where she has been ever since, and during which time she suffered from incontinence of urine.

Examination.—When admitted to the hospital she was obese, and fairly well developed. The tongue was protruded promptly in the median line and was slightly tremulous. Although both arms were moved fairly well, there was distinct loss of power on both sides, more apparent on the left. No note was made of the power in the legs. The right pupil was larger than the left, and both were moderately dilated. Both biceps tendon reflexes in the arms were increased. Knee-jerks were increased on both sides. There was no clonus, and stroking the plantar surface was followed by flexion of the toes. No loss of sensation could be demonstrated. Speech was somewhat thick, but the mentality was good. The abdomen was extremely prominent from two inches below the epigastrium to three inches above the pubes, not extending to the flanks. This mass was tympanitic, painful to pressure, and could not be reduced. Examination of the lungs was perfectly negative. There was no abnormality of the heart, except that the second aortic sound was accentuated. The urine showed a specific gravity of 1.033, a trace of albumin, epithelial cells and hyalin casts. The pathologic diagnosis was congestion of the kidneys and lungs. The patient died 14 days after admission.

Microscopic Findings.—Sections from the right and left paracentral regions, the medulla and the cervical cord were studied by the Marchi and Weigert hematoxylin methods, and stained by hemalum, eosin and thionin. Round cell infiltration of the pia and left paracentral region was found, and very slight thickening of the blood vessels, which was more marked on the right side. There was some perivascular distension in the cortex on both sides. In the left paracentral region there was a good deal of perivascular distension, but no evidence of any acute or old degeneration of the pyramidal tracts. By the Nissl stain there was no demonstrable disease in the nerve cells. No disease of the motor tracts was demonstrable by the Weigert or Marchi methods.

The lesions in this case were probably syphilitic. The perivascular distension in this case is worthy of note, as there can be no doubt that this condition, when widespread, causes some functional changes in the cortical nerve cells.

Case 4.—A. B., aged 71, a laborer, was admitted to the hospital in the service of Dr. Mills, Dec. 11, 1905. His was probably a case of bilateral hemiparesis. No previous history was obtainable.

Examination.—There was loss of power in both arms, greater in the right than in the left. Both legs were weak. The arms and legs were markedly emaciated. There was a paralysis of the right side of the face, and he was unable to speak clearly.

The pupils reacted to light and in accommodation, but vision was either entirely gone or greatly diminished. There was apparently no involvement of the motion of the eyeball. He could close both eyelids easily, the left better than the right. There was some weakness of the frontal portion of the occipitofrontalis muscle. The tongue was protruded toward the left and was tremulous. The pectoral muscles were wasted and were the seat of a fibrillary tremor. All the reflexes were exaggerated. Dorsal flexion of the toes of both feet followed stroking the plantar surface. The heart-beat was intermittent; the second aortic sound was scarcely audible. Moist, harsh râles were heard throughout the lungs but no other signs of disease were demonstrated. The specific gravity of the urine was 1,023; albumin was present and also hyalin casts, leucocytes, and a few epithelial cells. The patient died 9 days after admission.

Autopsy.—This was performed the following day. The pathologic diagnosis was croupous pneumonia, ulcerative endocarditis, primary cancer of the pancreas with secondary involvement of the liver, the right kidney and the mesentery; parenchymatous nephritis with interstitial change.

Microscopic Findings.—Sections from the right and left paracentral regions, the optic chiasm, the medulla, and the spinal cord from the cervical region, were studied by the Marchi method, and the Weigert hematoxylin method, and stained by hemalum, eosin and thionin. The pia in the right paracentral region was infiltrated with round cells. There was also slight infiltration of the pia surrounding the medulla. In the right paracentral region small areas of softening were seen. The blood vessels were numerous, and a few compound granular cells were observed. There was a moderate amount of pervivascular dilatation in the left paracentral region. No areas of descending degeneration were made out. The paracentral cortex, stained by thionin, did not reveal any cellular change worthy of mention.

This was a case of syphilitic disease of the nervous system, and exhibited in one section a small area of softening, together with the usual round-cell infiltration of the pia of syphilis.

Case 5.—L. R., a woman, aged 53, was admitted to the hospital, service of Dr. Spiller, June 6, 1904.

Examination.—Patient was brought to the hospital in a half-conscious condition, and when examined was muttering incoherently, responding slowly and indistinctly when addressed. She was apparently fairly well nourished, but showed signs of emaciation. She was unable to move the left leg in the slightest degree. The notes do not say whether there was any paralysis of the arms, but mention the fact that both arms were greatly emaciated, and both thenar and hypothenar eminences were flabby. The right foot was inverted to an extreme degree. The mouth was drawn to the right, but the nasolabial folds were of equal prominence, and she could wrinkle her forehead evenly on both sides. The tongue was pushed somewhat to the right, although she could not protrude it far beyond the lips. Both pupils were contracted and equal, but reacted to light in accommodation and convergence. Both eyeballs had good range of motion. There were no intraocular palsies. The reflexes were performed quickly and equally on both sides. The heart's action was regular; no adventitious sounds were heard, except that expiration was slightly prolonged at the left apex. The aortic and pulmonic second sounds were distinctly accentuated. There was incontinence of urine and feces. The lungs showed no abnormality. Specific gravity of the urine was 1,017; it contained hyalin and granular casts, besides epithelial cells, urates, and a trace of albumin. Patient died 12 days after admission.

Autopsy.—An anatomic diagnosis of bronchopneumonia of the left lung, general arteriosclerosis, and chronic interstitial nephritis was made.

Microscopic Findings.—Sections from the right and left paracentral regions, the pons, the cervical and lumbar cord, and the optic nerves and chiasm were studied by the Marchi method, the Weigert hematoxylin method, and stained by hemalum, eosin and thionin. In the pia of the left paracentral region there was a round cell infiltration. The blood vessels of the pia were markedly thickened. This was true also of the right paracentral region, where the change was slightly more marked. A moderate amount of perivascular distension was observed in the right paracentral cortex. The pia surrounding the medulla and spinal cord showed the same change as that seen in the cortical region, although it was not so intense. In a few Betz cells in the right paracentral region the nuclei were slightly eccentric without any other observable change. The Betz cells in the left paracentral region seemed to be intact. The cells from the lumbar and cervical enlargements appeared to be normal. There was no degeneration of the motor tracts in the medulla and spinal cord.

This was also a case of cerebrospinal syphilis, showing cellular infiltration of the pia, distension of the perivascular spaces of the cortex, and marked vascular changes, constituting lesions which satisfactorily explain the palsy present.

Case 6.—M. C., aged 66, was admitted to the hospital, service of Dr. Spiller, April 10, 1904. The family and previous histories were largely negative, excepting for the fact that an uncle had died of "fits," and that the patient had been a steady, although not excessive, drinker of alcohol.

She presented a history of fits when she was 2 or 3 years of age, which continued up to the time of her admission, recently at intervals of one to three months.

Examination.—There was no loss of power in the legs. The right pupil was larger than the left; the tongue was protruded straight. There was no facial atrophy. The station and gait were good. The grips were normal. The biceps, triceps, and knee-jerks were increased on both sides. There was no clonus, and no Babinski.

No further notes were made in this case until June 18, when the patient was found unconscious with all the limbs spastic, although forced movements were possible. The biceps, triceps, wrist, patellar and Achilles jerks were prompt and equal, and the plantar reflex was normal. There was distinct ulnar deviation of the left hand, the last phalanges being extended, the others flexed. The appearance resembled rheumatoid arthritis, and was present on both sides in the hands, and to a less extent in the feet. Patient lay with the thighs flexed on the abdomen, and the legs flexed on the thighs. The left pupil responded slightly to light. There had been incontinence of urine since the previous day. Until one week before the patient had been an attendant in the out-wards of Blockley, when she had what was called a "spell." She had been bedfast since this, in a semi-stuporous state. Examination of the urine revealed a trace of albumin, hyalin casts, leucocytes and epithelium. The specific gravity was 1,005. This condition suggested some irritation of the motor tracts, and was probably uremic.

Autopsy.—The pathologic diagnosis was chronic interstitial nephritis; hypertrophy of the heart; endocardial hemorrhage; arteriosclerosis; and chronic pleurisy.

Microscopic Findings.—Sections from the right and left paracentral regions, the medulla, and the cervical and lumbar sections of the spinal cord were studied microscopically. These sections were stained by thionin, hemalum and eosin, and by the Weigert hematoxylin and Marchi methods. The pia of the right paracentral region was slightly thickened, and moderately infiltrated with round cells. There were found also in the pia numerous red blood cells. In the left paracentral region the pia showed the same change, but it was slightly more thickened and contained a few more round cells. In the cortex of the left paracentral region a marked distension of the perivascular spaces of the small capillaries could be seen. This was also true on the right side, but to a less extent. The cells of Betz did not show any signs of degeneration, as shown by the thionin stain. No evidence of acute or chronic degeneration of the pyramidal tracts, either in the medulla or the spinal cord, could be demonstrated either by the Marchi method or the Weigert hematoxylin stain.

The only pathologic change of significance in this case, therefore, was the presence of thickened blood vessels and a marked perivascular distension, especially in the left paracentral region. A moderate round-cell infiltration of the pia suggested the possibility that this also was a case of specific disease.

CASE 7.—E. M., aged 65, a laborer by occupation, was admitted to the nervous wards of the Philadelphia General Hospital, service of Dr. Mills, Nov. 21, 1905. His mental condition precluded obtaining any previous history.

Examination.—On admission he was delirious, talking incoherently most of the time, although he occasionally appeared to comprehend when spoken to. The breath had a uremic odor. He was somewhat emaciated and very anemic. There appeared to be a slight loss of power in the left arm, which was also somewhat spastic. The left pupil was dilated and did not react to light, in accommodation or convergence. The notes state that he could not see out of the left eye. The right pupil was moderately dilated and reacted to light, in accommodation and convergence. The associated movements of the eyeballs were normal, excepting for a slight weakness of the external rotation of the left eye. Examination of the chest and abdomen was negative. There was a faint murmur at the aortic cartilage, systolic in time, and the second aortic sound was accentuated. Urinary analysis: Specific gravity, 1,020; albumin present; hyalin casts present; leucocytes and red blood cells. Clinical diagnosis was chronic interstitial nephritis, uremia, and endocarditis. Patient died 24 hours after admission.

Autopsy.—Hemopericardium, chronic interstitial nephritis, and beginning cirrhosis of the liver were diagnosed.

Microscopic Findings.—Sections from the paracentral regions, the cerebral peduncles, the optic chiasm, and the cervical regions were preserved and studied by the Marchi and Weigert hematoxylin methods, and stained by eosin, hemalum and thionin. In the right and left paracentral regions a moderate round cell infiltration of the pia was seen. This was also present in the pia around the medulla and pons. The blood vessels of the pia were markedly thickened, and in some places almost obliterated. The pia and blood vessels of the spinal cord showed a similar change. By the hematoxylin method degeneration was found in the right pyramid in the medulla, confined to the outer half. This degeneration was found also in the cervical region on the left side, and was diffuse and situated in the direct pyramidal tract on the left, and the crossed pyramidal tract on the right. There was no acute degeneration observable by the Marchi method. A small area of softening was found in the right lenticular nucleus, but otherwise the specimens showed practically no other abnormality.

This was a case of old lesion of the motor tracts, probably secondary to a necrotic process, of which one was found in one of the lenticular nuclei. Antemortem diagnosis of these lesions was impossible, and this case, a man of 65, illustrates how impossible it is to determine, without an autopsy, whether the paralysis occurring during a uremic state is due to syphilitic foci and softening, common in the old, or to some other cause the nature of which is at present speculative.

In the absence of previous history in this case it can not be stated whether there was any marked palsy apparent before the onset of the condition which was present on his admission to the hospital. Nor is there any way of determining whether this was a case in which an old lesion was awakened by the uremic state. The notes state that there was a slight weakness of the left arm, which was also spastic, and the probability is that this was a symptom that antedated the onset of the uremia. The diagnosis of uremic paralysis was entirely justifiable in this case, and only by a histologic study of the specimens could a correct diagnosis be made.

CONCLUSIONS.

I believe that the small areas of softening which I have described in this paper and elsewhere, are often the cause of hemiplegias or double hemiplegias, which have not been associated with any gross anatomic change. I wish also to emphasize the importance of careful and extensive histologic studies of the brain and spinal cord, which should be made before it is possible to exclude or determine the presence of demonstrable lesions in these cases.

Another cause for these paralyses which should not be overlooked is syphilis. I believe also that the perivascular distension, which is extensive in some of these cases, plays some part in the causation of the paralysis, probably by causing pressure on the cells of the cortex, a pressure which could come and go according to the amount of leakage from or absorption of the fluid into the perivascular spaces. This may be taken as a possible explanation of some of the cases of transient palsies with which every one is familiar as occurring in uremia and arteriosclerosis. I take this opportunity to express my sincere thanks to Dr. Charles K. Mills and Dr. William G. Spiller for the privilege of studying and reporting these cases.

The Connective Tissue Character of the Septa of the Spinal Cord as Studied by a New Stain

By ALFRED REGINALD ALLEN, M. D.

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THE CONNECTIVE TISSUE CHARACTER OF THE SEPTA OF THE SPINAL CORD AS STUDIED BY A NEW STAIN.*

By Alfred Reginald Allen, M.D.,

OF PHILADELPHIA.

FROM THE DEPARTMENT OF NEUROLOGY AND THE LABORATORY OF NEUROPATHOLOGY OF THE UNIVERSITY OF PENNSYLVANIA.

In working on this new method of staining nervous tissue, I have been able to demonstrate the fact that the posterior medium septum of the spinal cord, as well as a number of the septa of the lateral columns, contain connective tissue which is continuous with the pia. This statement is at absolute variance with the teaching that these septa are composed of exclusively neuroglia tissue.

In staining to demonstrate these pial septa the method is as follows:—

(a) The material should be hardened as if for staining by the Nissl method.

(b) Sections, after washing in distilled water, are placed in a 0.5% aqueous solution of azure II. (or azure I.) and steamed for two or three minutes and then allowed to cool for about fifteen minutes.

(c) Wash in distilled water.

(d) Place in absolute alcohol and gently agitate until clouds of blue cease to be given off.

(e) Place in a mixture composed of carbolic acid 20 toluol (boiling point 110°-112°) 80 for about one minute.

(f) Place in a solution of eosin (Grübler w. g.) in absolute alcohol (alcohol 60, eosin 0.03-0.05), and as soon as the change from dark blue to purple becomes pronounced, remove at once and—

(g) Place in carbol-xylol (25% solution of carbolic acid in xylol). Remove section from this before it turns pink, as after this the differentiation is greatly impaired.

(h) Place in pure xylol.

(i) Mount in xylol balsam or xylol damar.

The only difficulty that will be experienced by those using this stain for the first time will be the steps marked "f" and "g"

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*Read before the Philadelphia Neurological Society March 27, 1906.

Only practice will teach when to take the section out of the eosin-alcohol solution. I advise those wishing to obtain good results to pass through two sections, leaving the first in the alcohol eosin solution only five seconds and the second section about a minute, and then examining each to determine the faults of the two extremes.

In a properly stained section the pia is a vivid pink and the subpial neuroglia tissue blue. The tigroid substance of the anterior cornual nerve cells is well brought out, and the structure of the dendritic processes is particularly clear. There is a differentiation in the walls of the blood vessels, the intima and media staining blue, the adventitia pink.

When sections are hardened in Müller's fluid, the nerve cells loose their Nissl granules. If the material be first placed in formalin solution 5% to 10% for two or three days, and after that in Müller's fluid, the Nissl granules are preserved, but in this case the differentiation between the pia and the subpial neuroglia tissue is lost. The hardening in Müller's fluid, after the use of formalin, gives a very beautiful picture, in that it brings out the axis-cylinder processes, staining them blue. In this case the medullary sheath stains blue.

A longitudinal section of the spinal cord shows very clearly the collateral branches of the axis cylinders.

This stain does not give any better results with material kept too long in the hardening fluid than can be obtained with any other process.

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